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THE EFFECTS OF CHEMICAL SUBSTANCES UPON THE ELECTRICAL RESPONSES OF THE COCHLEA. I. THE APPLICATION OF SODIUM CHLORIDE TO THE ROUND WINDOW MEMBRANE*

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PRINCETON

The depressant effects of various chemical substances upon the action of living cells are generally known, and such substances are often used as a check on the physiologic basis of phenomena under observation. In experiments on the electrical responses of the ear, chemical agents have frequently been employed for this purpose.

Chemicals were first applied directly to the cochlea by Adrian, Bronk and Phillips.[1] In their investigation of the source and nature of the electrical responses they injected cocain solution through the round window membrane, and observed that the responses faded rapidly. The injection of physiologic saline solution had little effect. Guttman,[6] using the same procedure, found the responses to be abolished by cocain, quinin, salicyl and pilocarpin solutions, but not by saline, camphor and strychnin solutions.

Davis, Derbyshire, Lurie and Saul[2] placed novocain crystals on the surface of the round window membrane and thereby abolished

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the responses without the attendant mechanical damage of the injection procedure. Hallpike and Rawdon-Smith[7] reported a rapid and steady decrease in the responses after application of cocain or sodium chloride crystals.

The above observations were incidental to general studies of cochlear activity. A direct investigation of the effects of chemical substances upon the cochlear responses has been pursued by Fowler and Forbes.[3,4,5] They reported a progressive diminution in the responses obtained from dogs and cats after application to the round window of glycerin, sodium chloride, calcium chloride and quinin di-hydrochloride, and complete abolition if these substances were applied in sufficient quantities and left to act for a long enough time. There was little evidence of recovery of function over periods up to twenty-five days. No significant diminutions were found after application of distilled water, glucose and dextrose. Conditioned reflex tests on some of the animals confirmed in a general way the results of the electrical observations.

Fowler and Forbes reported that the diminution of responses was related to the frequency of stimulation: high tones were impaired earlier and to a greater degree than low tones. Histologic examinations of the ears revealed marked destruction of hair cells and sometimes of other tissues; this destruction usually was most prominent at the basal end of the cochlea, and grew progressively less toward the apex. The greater impairment of high tones, in relation to the locus of destruction, was regarded as supporting a place theory of hearing.

The present paper reports the results of a quantitative study of the effects of sodium chloride upon the cochlear potentials of the guinea pig.

APPARATUS AND METHOD

The stimulating apparatus consisted of an electric oscillator, filters, attenuators and a loudspeaker, as described previously.[9] The tones were of a high degree of purity and of known intensity. After the bulla was opened from the posterolateral side, a silver foil electrode was placed on the round window membrane. An indifferent electrode of silver wire was inserted in the neck muscles. These electrodes conducted the cochlear potentials through an amplifier to a cathode-ray oscillograph. A calibration of the amplifier and oscillograph permitted a determination of the magnitude of responses at the round window in absolute units.

After measurement of the responses under normal conditions, a small quantity of sodium chloride in the form of crystals was placed

on the membrane of the round window. Further measurements were made immediately thereafter, and repeated at frequent intervals over periods which varied in different experiments from 20 minutes to 3½ hours. Most of the tests were made with the frequencies 300, 1000 and 7000~, but sometimes additional tones were used. The procedure was varied in certain experiments, as described below.

The quantity of salt that was applied to the membrane varied in different experiments, but usually was about three crystals. This paper reports the results of experiments on twenty ears.

RESULTS

Figs. 1-3 present results that are typical of those obtained by the procedure described above. In these experiments the intensity of stimulation was adjusted to give a response of approximately 100 μv under normal conditions for each frequency used. The "standard" intensities thus obtained were employed throughout the series of tests. Time in minutes is shown on the abscissa, and magnitude of response on the ordinate. Two ordinate scales are given, one on the left for absolute values of response in microvolts (μv.), and one on the right for the amount of change in decibels (db.) referred to a standard value of 100 μv.

Fig. 1 shows the results of two experiments in which the responses were measured at intervals of about 20 sec. for a single tone. The curve for 1000~ is for one ear, and that for 7000~ for the other ear of the same animal.

Fig. 2 shows the results of an experiment in which tests were made on the same ear with three tones, 300~, 1000~ and 7000~, presented in rotation. The points on the curves to the left of the zero line represent normal responses, and those to the right the responses after the application of salt. Fig. 3 shows similar results on another animal.

Our results agree with those of other investigators in showing an impairment of cochlear responses after the application of sodium chloride. In certain respects, however, our observations differ from those previously reported.

Initial Augmentation.—When measurements were made with a minimum of delay after the application of salt, the responses in most cases showed a temporary increase in magnitude. Typically, this augmentation appeared within about one minute after the application and reached a maximum in two to five minutes, after which the responses rapidly declined.

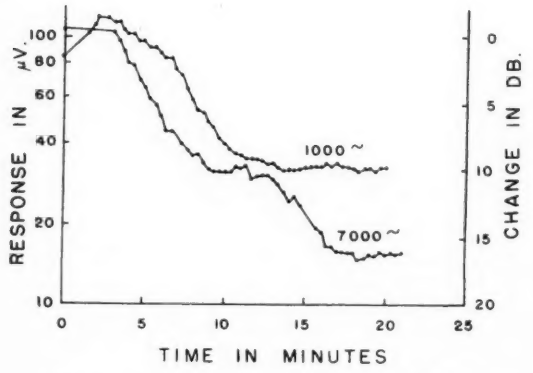

Fig. 1. Effects of sodium chloride on the cochlear responses to 1000— (left ear) and to 7000— (right ear) in guinea pig No. D 7. The measurements at zero time are for normal conditions, before application.

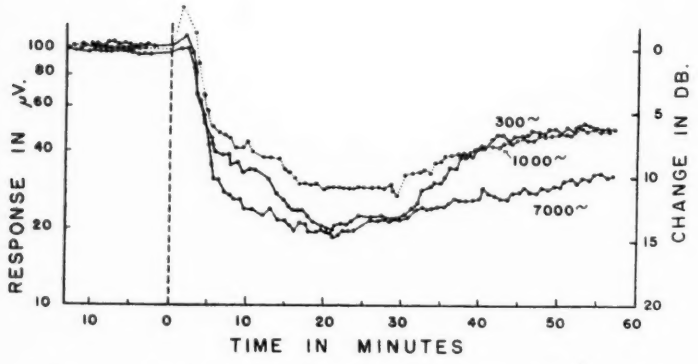

Fig. 2. Effects of sodium chloride on responses to 300, 1000 and 7000—, measured in the same ear. Normal responses are shown to the left of the zero line. Guinea pig No. D 17 L

If the electrode was placed on the bony rim of the window, or elsewhere on the bony wall of the cochlea, the initial augmentation was very prominent.

Regularity.—The degree of regularity of the responses can be observed in curves such as those of Figs. 1 and 2, in which the measurements were made at frequent intervals. Some of the smaller variations were due to observational error, and many of the abrupt changes were probably caused by protective reflexes (contractions of the tympanic muscles). But the more regular changes represent variations of sensitivity which we consider to be due to alterations in the physiologic state of the sensory cells. Variations in the rate of decline were common.

Degree of Impairment.—The reduction of responses is a function of the quantity of salt applied. When only about three crystals were used, as in most of the experiments of this series, the curves leveled off after a drop which varied in different cases from 10 to 30 db. When as many as ten to fifteen crystals were used, the responses dropped rapidly until they could no longer be measured—i. e., dropped something more than 50 db. below their original level. When one application had caused an impairment that after several minutes seemed not to be proceeding further, a second application produced a further decline.

Recovery.—Some of the curves showed recovery, at least of a temporary kind. This phenomenon is illustrated in Fig. 2, and is especially prominent in the curve for 1000~ in Fig. 3. In this curve is shown a marked rise of responses from a level of 3.8 μv. to a level of about 10 μv. Similar recoveries, usually smaller in degree, were of frequent occurrence. In most cases they were followed by further decline.

Relation to Frequency.—As mentioned above, Fowler and Forbes reported that in most of their experiments "the elimination of the cochlear response apparently took place earlier in the higher frequencies than in the lower." The results of the present experiments, however, can not be formulated in so simple a manner.

The curves of Figs. 2 and 3 show that initially the responses fell rapidly for all tones and at approximately the same rate. After a few minutes the rate of decline became less, and typically there appeared a differentiation of frequency: the low tones tended to level off sooner and to a greater extent than the high tones. Consequently, after a sufficient time the various frequencies were differentiated in the amount of response for a corresponding intensity of stimula-

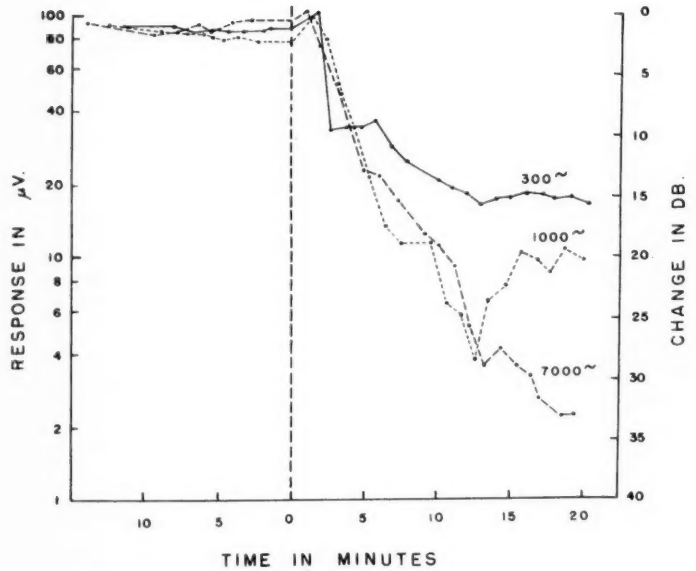

Fig. 3. Effects of sodium chloride on responses to 300, 1000 and 7000~, measured in the same ear. Normal responses are shown to the left of the zero line. Guinea pig No. D 9 R.

tion. Usually this differentiation was in order of frequency, but exceptions occurred.

When the quantity of sodium chloride was greater than that used in the cases illustrated, the decline was more rapid in rate, and it was correspondingly more difficult to follow the courses of the several tones with precision. But so far as we could determine the same relations held: all tones fell together in the beginning, but after a time the high tones usually fell at a more rapid rate. It may be mentioned in this connection that some cases of Fowler and Forbes showed general impairment rather than an impairment in order of frequency.

Intensity Functions.—In a number of experiments the measurements, both before and after application of sodium chloride, were made at various intensities of stimulation. This procedure revealed the functional relations between stimulus intensity and magnitude of response under normal conditions and at various stages of impair-

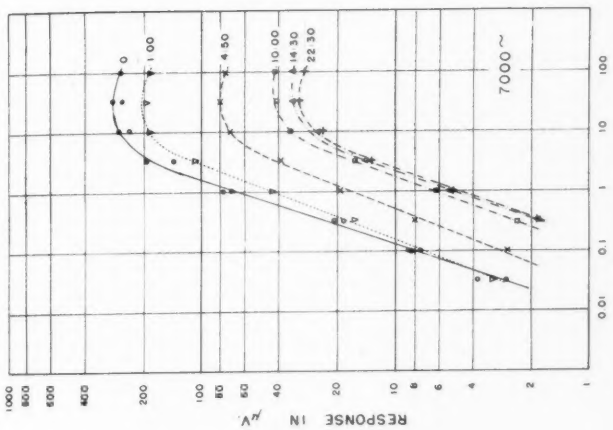

Fig. 5. Intensity functions for 7000~ before and at intervals after application of sodium chloride. The time following application is shown in minutes and seconds at the right side of each curve. Guinea pig No. D 5 L.

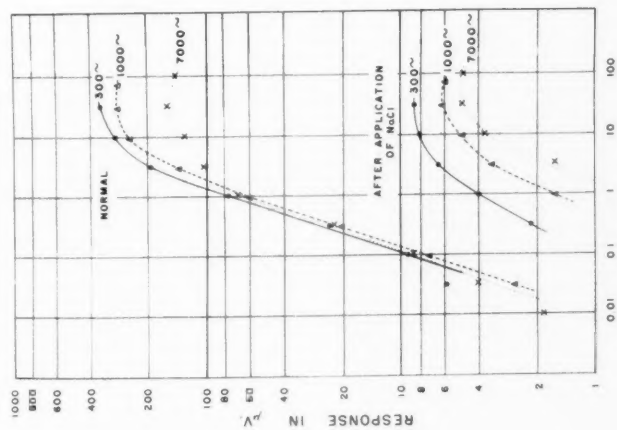

Fig. 4. Intensity functions for 300, 1000 and 7000~ before and 47 to 50 minutes after application of sodium chloride. Guinea pig No. D 6 L.

ment. Typical results of these measurements for different frequencies are shown in Figs. 4 and 5.

Fig. 4 shows intensity functions for 300, 1000 and 7000~ before and after application of salt. In this figure the abscissa indicates sound intensity in units of pressure (bars, or dynes per square cm.), and the ordinate indicates the magnitude of response in microvolts. The fact that the three normal curves are practically coincident throughout their main course indicates that the animal's sensitivity was nearly equal for these tones. The smaller curves to the right represent the functions that were observed 47 to 50 minutes following the application of salt. The loss of sensitivity is shown by the downward displacement of the curves. Another feature to be noted in this figure is the change of slope after the application of salt. The slope was reduced for all three tones, but the amount of reduction seems related to frequency: it was greatest for 300~, and least for 7000~. This relation between frequency and change of slope is typical for high degrees of impairment.

Fig. 5 shows, for another animal, a series of functions for a tone of 7000~, obtained at intervals after the application of salt. The number at the top of each curve shows the time in minutes and seconds after application of salt. The curve marked "O" represents the normal function before the application. The figure shows a progressive decline of responses, but with only slight alterations in form and slope of the functions.

In some of the animals the functions, after application of salt, showed unusual irregularities, especially those for tones below 1000~. The cause of these irregularities is uncertain.

Observations From the Apex.—In a few animals the procedure was varied. (1) In some the bulla was opened from the ventral as well as from the posterolateral side, and an active electrode was placed on the bone at the apex of the cochlea in addition to the one on the round window membrane. Responses were observed alternately from the two positions before and after application of salt to the membrane. (2) In other animals the two active electrodes were placed on apex and round window as described, and the salt was applied at the apex through a minute opening that had previously been made. With the latter procedure it was necessary to observe the effects of the operation of opening the cochlea before proceeding with the application of salt.

These experiments have been too few in number for final report, but preliminary results from both procedures were as follows.

There was a differentiation of electrical activity as recorded from the two positions.[7] After application of salt to the round window, the responses which were picked up by the round window electrode were impaired sooner, and the impairment was more severe, as compared with those picked up by the apical electrode. After application of salt to the apex, the responses from the apex showed first a marked augmentation, then a rapid decline; the responses from the base showed little change until after those from the apex had suffered a considerable loss.

DISCUSSION

The results of these experiments do not lead to any simple theory of the effects of sodium chloride upon the electrical responses of the cochlea. Several features deserve particular consideration.

1. The initial augmentation that was revealed in those cases in which measurements followed promptly after application of salt can most easily be explained as due to an increase in the electrical conductivity of paths between the active electrode and the sources of potential.

2. The early, rapid decline of responses for all frequencies admits of no ready explanation. It does not seem likely that within the brief times involved the substance could diffuse to all parts of the cochlea so as to affect all active cells equally. We incline, therefore, to some such hypothesis as a general alteration of pressure in the cochlea as the result of the action of sodium chloride.

3. The further decline of responses, in which there often appeared a systematic relation to frequency, is more readily explained on the basis of the "tentative hazard" of Fowler and Forbes of a change of permeability of hair cell membranes, or of some other impairment of the sensory cells.

4. The frequent appearance of partial recoveries suggests that the above processes are in some degree reversible.

5. The variations in the rate of loss after application of salt are perhaps due to the complexity of the paths of diffusion through the cochlear canals, and to the irregular operation of processes of recovery.

Relation to Cochlear Localization.—Fowler and Forbes regarded their results as in support of a place theory of hearing. Their observation of a progressive loss of frequencies from high to low was related to the expectation that a substance in its diffusion from the

round window would affect first the basal elements and then the middle and apical elements in turn.

As mentioned above, our results do not indicate any single, simple process in the action of sodium chloride, and therefore do not readily fit the theoretical pattern outlined by Fowler and Forbes.

A consideration of the early general loss of responses leads to these alternatives:

If we can postulate a prompt and at the same time a general action of the substance on the cochlea, as has been attempted in the preceding section, a theory of cochlear localization is still tenable. But if the action of the salt is strictly local, and the diffusion rate is of the order to be expected, the alternative would seem to be the absence of cochlear localization.

That some localization exists in the cochlea, but that it is broad rather than restricted, is indicated by the observation that an electrode on the round window membrane does not reveal changes of exactly the same form as one on the apex of the cochlea. The inference is that the two electrodes record effects which to some degree reflect the action of different cells in different places.

An explanation of the forms of the intensity functions after application of salt can be consistent with this hypothesis of broad cochlear localization. Our reasoning, which like most of the foregoing, takes the form of "tentative hazard" (to borrow Fowler and Forbes' phrase), is as follows: The normal relation between stimulus intensity and magnitude of response in the guinea pig is a power function with an exponent that is close to unity, as discussed elsewhere.[9] This function is probably based upon two concomitant processes. An increase in sound intensity probably occasions (1) an increase of activity in cells already involved, and (2) an extension of the region of response to bring in new cells. The regularity of slope of the curves suggests (though it does not necessitate) that both processes bear a simple relation to intensity. There is no reason to suppose that the application of salt is followed by a change in the extent of response for any given stimulus, and therefore the observed decrease of response is to be attributed to a reduction in the activity of some or all the cells which serve the frequency in question.

The change in slope shown by the curves can be attributed either to an alteration in the form of the intensity function for individual cells or to a difference in the impairment of cells at different places. The latter hypothesis seems preferable in view of the fact that the curves for high tones show less change in slope than those for low

tones, and the fact that local degenerations are found histologically.[5] For the high tones the effects should be fairly uniform over almost their whole region of action if they are broadly localized at the base where the diffusion should be most general. For the low tones, however, the effects should be greater on the basal border of their region of action, and as this border area is brought into play in the extension of range for greater intensities the contribution to the response should be below the usual proportion.

SUMMARY

A series of twenty experiments on guinea pigs dealt with the effects of sodium chloride upon the electrical responses of the cochlea. The magnitude of responses was determined in absolute units for stimulating tones of known intensity, both before and after the application of sodium chloride. In most of the experiments the substance was applied to the round window membrane; in certain others it was applied through an opening at the apex of the cochlea.

Results are given to show the course of changes in the responses for different frequencies subsequent to the application of sodium chloride. Typically, there was an initial augmentation, followed by an early rapid loss and then by a protracted decline of more moderate rate. The early loss occurred in about the same degree for all frequencies. The secondary loss was usually differentiated with respect to frequency, with high tones commonly suffering more than low tones. In some cases there were partial recoveries.

A study of the functional relations between intensity of stimulation and magnitude of responses showed little alteration in form or slope of functions for high tones, and slight reductions of slope for low tones.

Concurrent observations from round window and apex showed a difference in the effects of sodium chloride, as recorded from the two positions: the impairment of response was greater from the electrode in the region where the substance was applied.

Several processes are suggested in explanation of the results. The early, rapid loss, which involved all frequencies, is attributed to some general process such as a disturbance of pressure relations within the cochlea. The secondary decline, in which high tones usually were involved more severely than low tones, is regarded as consistent with Fowler and Forbes' theory of a progressive impairment of hair cells.

Three features of the action of sodium chloride are discussed in relation to the problem of cochlear localization. These are the

low degree of specificity as regards stimulus frequency, the differences in the observations at base and apex of the cochlea, and the greater reduction in slope of the intensity functions for low than for high tones. It is suggested that these facts indicate broad rather than specific localization of tones in the cochlea.

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XXVIII

THE DEFENSE MECHANISMS OF THE UPPER RESPIRATORY TRACT*

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Nearly forty years ago the late Henry Wagner addressed this association on the natural immunity of the mucous membrane of the respiratory tract, maintaining that it would eventually be found that immunizing effects depend not on the mechanical protection of mucin but upon the "protoplasmic activity" of the living body cells; and he drew attention to the production of "protective enzymes" by the leucocytes.[1] Following his prophetic vision, as interpreted in 1910 by Birkett[2] and later by Coates[3] and Halsted,[4] we found many lacunæ in the theory and practice of autogenous vaccination against sphenoethmoidal sinusitis, reported to you in 1925.[5] Variability in the immune properties of respiratory membranes, especially from the standpoint of their unstudied content of reticulo-endothelial structures capable of stimulation, multiplication and phagocytosis,[6,7] directed us in subsequent years to examination of the characteristics of such membranes as disclosed by vital staining, and thus to the elaboration of technic for research upon living membranes in experimental animals.[8] Through the generosity of the research committee of the Council of the American Academy of Ophthalmology and Otolaryngology this work has been continued for six years past,[9 to 16] and has led to an investigation not only of the normal anatomy of these structures,[8,10] the effects of various solutions,[9,11,12] and of medicaments and therapeutic procedures upon sinus membranes,[9,11,12,13,16] but also of the lymphatic drainage of the sinuses and pharynx in relation to the lung,[11,12,15] and of the autonomic innervation and pathways for reference of pain from the nasopharynx.[14]

Brief recapitulation of these results will suggest certain clinicopathologic conclusions as at present aligned with other and similar

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research. Our study has been primarily anatomic rather than immunologic, and we have not therefore investigated those factors which depend upon circulation of immune bodies in the blood plasma, or biochemical factors except as to surface application. Such factors, general to the entire body, include allergic and other special sensitizations, also familial and constitutional details, and touch upon the whole question of natural and acquired immunity.[6,7]

The respiratory tract serves the primary functions of bringing air to the pulmonary capillaries and of carrying carbon dioxide to the exterior. The inspired air must be prepared, so far as possible, for contact with the moist and delicate membranes of the interior of the lungs, by passing it over the turbinate processes of the nasopharynx, and thus the temperature and moisture content of the inspired air is modified to conform more nearly to that of the body. Air laden with dust or containing bacteria is also cleansed to a large extent in the nasal passages.

MUCUS SECRETION

The chief mechanism by which this is accomplished is undoubtedly the covering of mucous secretion on the epithelium, which is produced by the goblet cells and the glands of the mucous membrane. Dust particles and no doubt bacteria, are caught by this secretion. It has been shown by Arnold, Ostrom and Singer[17] that 90 to 95 per cent of viable bacteria are inactivated in five to ten minutes when placed on the nasal mucosa. Both inert dust particles and inactivated bacteria are removed, with the mucus, by the action of the cilia of the epithelium, aided occasionally by sneezing. The coating of mucous secretion must therefore be regarded as the barrage laid down by the epithelium against irritation, mechanical, bacterial and also chemical. Since mucous glands and goblet cells are found not only in the walls of the nasopharynx but also in the lungs, this protection is important throughout the respiratory passages. Mucous glands are found within the lung only in the larger bronchi, but goblet cells extend as far as bronchioles of 0.4 mm. diameter.[18] Cilia are found even more distally, being present on some of the cubical epithelial cells of respiratory bronchioles of 0.3 to 0.4 mm. diameter.

CILIA

The action of the cilia in the nasopharynx and accessory sinuses has been studied by Lucas,[19] Hilding[20] and many others. This is clearly a purposeful activity, designed to keep a fresh sheet of protective mucous secretion over the surface of the epithelial cells. Within the lung the same function is undoubtedly served, but with greater

difficulty because the cilia must work against the force of gravity. The accumulation of plugs of mucus in larger bronchi which would tend to block tributary bronchi of smaller size is guarded against by nerve terminations placed at the strategic points where bronchi divide.[21] These afferent nerve endings, when stimulated, produce reflex bechic blasts,[22] expelling the mucous secretion.

The entrance to the nasal chambers is lined with stratified squamous epithelium, with no covering of mucous secretion. This epithelium is a continuation of the skin and is very resistant to injury. The smaller respiratory bronchioles, alveolar ducts and air sacs of the lung likewise have neither cilia nor protective mucous secretion. Save for the considerable protection afforded by phagocytic cells, to be described below, the respiratory surface of the lung is exposed to any invading organism or noxious substance which has succeeded in running the gauntlet of the air passages to this point.

Increased secretion of mucus, under conditions of irritation or infection of any part of the respiratory tract where such secretion can be produced, is an attempt by the body to increase the protective mechanism by strengthening the first line of defense. The effect is partly chemical, no doubt, and partly mechanical. This attempted defense may be overdone, with resulting blocking of nasal passages, filling of accessory sinuses, and other manifestations which add to the burden of the organism and may set up vicious cycles.

Ciliary damage is caused by hypertonic, viscid or oleaginous solutions, slowing or stopping the ciliary beat and thus interrupting the normal flow of mucus.[8,9] Such delay may, however, be of temporary therapeutic advantage by permitting the volatilization of dissolved medicaments.

We were able to confirm, in 1934,[12] the prophylactic effect of solutions of tannic acid and of sodium alum against sinus membrane infection, paralleling its use against virus invasion of the meninges. This effect is due to temporary coagulation of mucus and cilia into a firm protective film through which germs and viruses cannot penetrate.

EPITHELIUM

The main part of the first line of defense is formed by the epithelium itself. The studies of Hilding[20] have shown that there is an adaptation of the epithelial lining in response to changes in ventilation in the nasal passages. A modification to stratified squamous epithelium, similar to that at the anterior nares, followed overventilation in his experiments. An increase in number of goblet cells

resulted when air currents were reduced. In the main respiratory passages the epithelium is very resistant and infection is rare. The resistance appears due to the protective effect of the mucous secretion and to the vigorous epithelial cells, which occur in many layers. Some of these cells extend to the tunica propria, basally, and have active cilia distally.

PARANASAL SINUSES

The paranasal sinuses differ from the main respiratory passages in being lined with a simple columnar epithelium which is ciliated and has relatively few goblet cells normally. The epithelium thickens enormously and the goblet cells increase in number under conditions of inflammation caused by infection. This modification must be considered as an adaptation of the epithelium and its mucous product to resist the infection. Normally the sinus linings appear to be less resistant to infection by reason of the poorly developed first line of defense. Added to this is the protection afforded invading organisms, once they gain entrance, by the structure of the sinuses.

The respiratory membranes rest on a tunica propria. In the nasal passages the connective tissue of this layer attaches the epithelium to rigid walls. There is no movement of muscle to force the lymph containing toxic substances along lymphatic channels or otherwise to help remove noxious elements from the membranes. Such removal is therefore slow, and noxious substances, once they have gained entrance into the mucosa, tend to accumulate. In the paranasal sinuses this condition is aggravated by the very thin tunica propria, which here is continuous with the periosteum of the rigid walls. With no great pressure behind and no movement of the sinus walls, toxic products accumulate. Lymphocytes and neutrophile leucocytes come to such places by migration from the blood stream.[8,9] There is also infiltration by fluids. The result is edema and a greatly thickened membrane filled with cells. The leucocytes very early begin to phagocytose bacteria and thus to fight off the invaders. The neutrophiles appear to be more successful elsewhere against pyogenic bacteria than against certain others, especially the tubercle bacillus.[23]

Contained within the normal tunica propria, also, are found scavenger cells, the histiocytes, which by proliferation or migration or both processes, increase rapidly in number in early stages of infection of sinus membranes.[8 to 13] They begin to resist the invading organisms by engulfing them, supplementing the efforts of the neutrophiles. There is evidence that even against certain types of pyogenic bacteria the histiocyte is more effective than the neutrophile leucocyte.

These cells, the neutrophiles and the histiocytes, or macrophages, constitute the second line of defense. In experimental infection with streptococcus in cat sinuses both types of cells appear in the infected membrane in great numbers during the first seventy-two hours.[9] The membrane becomes very greatly thickened. The neutrophiles, and sometimes the histiocytes, migrate into the epithelium and through it escape into the sinus cavity. The membrane appears to be a battleground. The thickening of the membrane, when it occurs at the ostia, closes the latter and by preventing drainage aggravates the condition within the sinus. In experimental cat sinuses the height of the acute infection is reached at about seventy-two hours. Following this the leucocytes largely disappear, the histiocytes become reduced in number, and another type of cell, namely, the plasma cells, gradually becomes predominant as the membrane subsides into what we have called a chronically infected membrane—that is, one which has not entirely cleared up in about one month.[8 to 13] The membranes are reduced in thickness from the swelling of the acute stage, but they still show considerable edema. The epithelium, when not destroyed, remains thick with numerous goblet cells. Patches of lymphocytes, in nodule like formation, are frequent. The latter phenomenon has never been observed in our normal cat sinus membranes.

PHAGOCYTES AND PLASMA CELLS.

The phagocytic functions of neutrophiles and of histiocytes are established and need not here be discussed. The function of the plasma cells, which follow these as an army of occupation in the membranes, is not well known.[9] Likewise the lymphocytes, which are among the first cells to reach an injured region by migration from neighboring capillaries, are of unknown function, in spite of numerous attempts at interpretation. Many histologists in recent years have adopted the view that lymphocytes give rise to plasma cells and other cells in the tissues, among other possible functions. However, on the basis of study of human bone marrow obtained from the living individual by sternal puncture, Osgood and Hunter[10] hold that plasma cells represent a distinct strain of leucocytes, not related to lymphocytes. If this view is correct the presence of plasma cells in the "chronic" sinus membranes, in the large numbers encountered by us, must indicate a very considerable migration into the membranes, since mitosis is very infrequent in the tissues and the number of plasma cells in normal sinus membranes is negligible. The presence of numerous plasma cells in infected sinus membranes can be interpreted in terms of function either as elaborators of defensive substances or as cells which absorb and then dispose of various products of tissue metabolism. (Schaffer, 1910, cited by Bunting.[23]) There is no indi-

cation that plasma cells phagocytose bacteria, but in chronically infected membranes there are considerable numbers of histiocytes which continue their phagocytic activity.[9 11 12 13]

From the clinical standpoint, we find that sinus membranes are materially damaged as to epithelium by viscid and oily solutions,[8 9] and by hypertonic solutions of magnesia, calcium and a considerable number of antiseptic compounds, including merthiolate,[12] mercurochrome,[12] scarlet red and oxyquinoline,[10] dichloramin,[12] azochloramid[13] and trinitrophenol.[12] Bactericidal power does not prevent epithelial irritation. Even normal saline solution becomes an irritant if used repeatedly, removing normal mucus and causing edema.[13] Amniotic fluid, while irritating to epithelium, tends to increase repair through round cell infiltration.[13] Mucosal congestion is increased by diathermy and by application of acetylcholine solution,[13] and reduced temporarily by ephedrin-resembling drugs.[13 16] Therapeutic use of the roentgen ray, which does not damage epithelium, destroys round cell infiltration and is followed by fibrosis but has no effect on existin infection.[11 12 13]

Modification of cellular components of sinus membranes is variously produced experimentally. Acute exacerbation of inflammation in chronic membranes is produced by solutions of histamine and acetylcholine.[13] Reparative changes in the tunica propria, with mobilization of histiocytes, are produced by milk of magnesia,[9] by calcium hydroxide, lactate and gluconate,[11] and by amniotic fluid.[13] The congestive effect of diathermy also brings in more histiocytes.[12] Certain ephedrin-resembling drugs seem to increase this reparative effect.[13]

Edema of the tunica propria is increased by solutions of sodium and chlorine,[9] reduced by calcium and magnesium.[9 11]

Marked increase in plasma cell infiltration and in fibrosis is observed with calcium and magnesium solutions.[9 11] and after diathermy.[12]

EUSTACHIAN TUBE

The eustachian tube, connected with the nasopharynx, is lined near the pharyngeal opening with ciliated pseudo-stratified epithelium. Its mucosa in this region is very similar to the sinus mucosa save that it is much thicker and contains more lymphoid tissue, the "tubal tonsil". Histiocytes are present.[10] The end of the tube which opens into the tympanic cavity has a thinner mucosa, lined with low columnar epithelium, still ciliated. This gives way at the tympanic cavity entrance to flattened epithelium without cilia. Histiocytes are

present in the tunica propria in this part of the tube also. Mucus secreting goblet cells are present only at the pharyngeal opening. Protection of the auditory tube against infection from the pharynx must therefore be afforded first by the mucus at its opening, then by the epithelium and its cilia, acting as a mechanical barrier, and finally by the phagocytic histiocytes

VASOMOTOR EFFECTS

The effect of edema and thickening of the sinus membrane as a factor in closing the sinuses and aggravating acute or residual infection is a fact of every day experience. Perhaps it is not so generally recognized that swelling of the membranes, producing closure of the ostia, may be brought about by dilatation of the mucosal blood vessels. These vasomotor changes are due to reflexes carried by the autonomic nervous system.[14] There is both clinical and experimental evidence that such changes are related to vascular congestion in the abdomen and the pelvis. The pathway involved, whether sympathetic or parasympathetic, is not clear and requires further study. Treatment to relieve the cause of abdominal or pelvic congestion is sometimes more important than local treatment of the sinuses.

Vasodilatation of extreme degree was produced experimentally by cervical sympathectomy, in experimentally infected sinus membranes, most severe in acutely inflamed tissues,[12] but also exceedingly well marked in tissue which had reached the stage of chronic inflammation with edema and plasma cell infiltration.[13]

LYMPHATIC DRAINAGE

The lymphatic drainage from the nasal cavity is to the superior cervical and the retropharyngeal lymph nodes. The lymph channels of the sinuses are small and difficult to demonstrate by injection with the usual gelatine mixtures.[12 13] When the sinuses, e. g., the frontal in the cat, are filled with india ink suspension[26] or with trypan blue solution,[15] the membrane having been injured to facilitate absorption, the ink or the dye soon appears in the retropharyngeal and the superior cervical lymph nodes. The trypan blue is always found, under these conditions, as particles contained in phagocytic cells, histiocytes and reticular cells. Its presence in the latter, which could not possibly have migrated through the lymph channels from the sinus membranes, indicates that the trypan blue, in solution, was carried through these channels to the lymph nodes. Dye-laden histiocytes are found in large numbers in these experiments, also in the sinus membranes.

Bacterial infection follows the same route. The retropharyngeal and superior cervical lymph nodes of cats whose sinuses have been infected with large doses of streptococcus are swollen and their cells unusually active. Phagocytic cells containing granules which, on the basis of staining methods used and morphologic appearance, must be interpreted as streptococci, are numerous. There are apparent changes in the staining qualities of these granules which must be indications of chemical modification. This is tentatively interpreted as due to effects of substances produced in the lymph nodes upon the invading bacteria. Maximow[24] states, on the basis of Marchand's work: "It is very probable that the defense function of the lymph nodes, especially their capacity of reducing the virulence of pathogenic micro-organisms, is partly due to the activity of the lymphocytes." The cervical lymph nodes must be regarded as the third and major line of defense in sinus infection, a defense which is designed to protect the rest of the body from the results of the broken down local defense of the membranes themselves.

When this line of defense breaks down as it occasionally does, especially in children, the body faces a serious problem. The infectious organisms are now released from relatively narrow channels adapted for strict control, to find their way to other parts of the body by various routes.

From the retropharyngeal lymph nodes directly there is possibility of release into the surrounding tissues. The local histiocytes become active phagocytes, and tissue reactions begin in an attempt to wall off the invaders by formation of fibroblasts and connective tissue.[15] If the phagocytes are overwhelmed but the tissue quarantine process is rapid enough a localized abscess may be the result. If the cause of the infection is not stopped locally in the retropharyngeal region it may continue downward in the tissue spaces between the esophagus and the prevertebral fascia to the upper mediastinum. This is an anatomic possibility, but is probably rare because of the distance involved and the very numerous phagocytic cells along the route, with numerous opportunities for sufficient tissue reaction to localize the invading organisms.[15]

Efferent lymphatics from the retropharyngeal nodes drain, however, into the deep superior cervical nodes. These constitute a most important part of the main third line of defense. The retropharyngeal nodes can be considered as outposts of the deep cervical nodes. The large deep cervical nodes usually are able to cope with the situation, but if they also break down, the drainage is into the cervical lymph ducts. These drain into the thoracic duct or the right lymph

duct, and so into the great veins tributary to the superior vena cava. The efferent drainage from the cervical lymph nodes thus reaches the right heart, and from this point the pulmonary capillary bed.[12] Trypan blue in this situation diffuses through the endothelium of the capillaries and is taken up by the phagocytic septal cells. These cells, lining the septa of the air sacs, are regarded by many histologists as identical with histiocytes elsewhere. At any rate, they are phagocytic. Some become detached, and as "dust" cells are found in the air sacs. The perivascular and peribronchial lymphatics become filled with dye-laden macrophages.[15] The lymph follicles in the bronchial walls show increased size and numerous dye-laden phagocytes, indicating migration of such cells from the outer portion of the lung through the lymphatics which drain into the pleural lymphatic network described by Miller.[13] Both intrapulmonary and pleural lymphatics drain into the bronchial and tracheobronchial lymph nodes.

THE LUNG

To what degree the pulmonary capillary bed constitutes a barrier to particulate material in the blood stream is problematical. Certainly there is no such barrier as is afforded by the Kupffer cells of the liver sinusoids. Trypan blue, introduced by the route described, however, is largely taken out of the circulation in the pulmonary bed. Sections of liver and spleen, which organs would eventually receive and phagocytose any material which escaped into the general circulation, show comparatively few dye-laden cells, although some are present.

The pulmonary bed, the bronchial lymph follicles, and especially the bronchial and tracheobronchial lymph nodes, thus constitute together a fourth barrier to material from the upper respiratory passages, if such material succeeds in getting beyond the superior cervical lymph nodes.[15]

Material that enters the trachea and lungs directly, as by dripping from the upper passages or with the inspired air, meets the phagocytic septal and dust cells in the air sacs without need of passing through the capillary endothelium. Septal and dust cells constitute the first line of defense in this case.[15] The question of epithelium in the air sacs is left out of consideration, since students of the lung are disagreed as to its presence. The further pathway of material reaching the air sacs directly would be the same as described above after such material is phagocytosed or gets into the lymph stream. Some of it, to be sure, would be expelled through the air passages with mucus and dust cells discharged with mucous secretion. The goblet cells and ciliated epithelium, described by Miller,[13] in the respiratory

bronchioles and larger air passages undoubtedly serve the same protective function here as they do in the upper respiratory tract. Material that gets into the air spaces themselves, however, has no such barrier, or only a single layer of flattened cells between it and the phagocytic cells, and must contend with the guard of phagocytes, as described.

To what extent bacterial invasion may follow the pathway, beyond the superior cervical lymph, described for trypan blue solutions, has not as yet been determined experimentally. Also the response of the cells within the lungs, especially of the capillary endothelium, to bacterial invasion must be determined before too close an analogy can be drawn between the reaction of ultramicroscopic colloidal nontoxic particles and that to toxin producing bacteria of relatively large size. It can only be said with assurance that from the sinus membranes to the deep superior cervical lymph nodes the pathway and general response to the two types of introduced substance is the same, save that the bacteria set up inflammatory processes not produced by the dye.

These observations, which have been somewhat dogmatically presented, inspire the hope that future studies in this field may be found to interrelate the problem of upper respiratory immunity not only with the various lines of defense here mentioned, but especially with the autonomic nervous system and its influence upon biochemical and endocrine factors. These regions form the gateway to the body; though well defended, they contain so many byways, with relatively poor protection in the human species, that every effort must be made to promote "the protoplasmic activity" postulated by Henry Wagner so long ago.

806 Medical Arts Bldg.

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XXIX

A NEW METHOD OF RADIUM APPLICATION IN CANCER OF THE BRONCHUS*

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AND

CLYDE K. EMERY

LOS ANGELES

The present day treatment of carcinoma of the tracheobronchial tree consists essentially of four therapeutic measures—surgery, surgical bronchoscopic diathermy, exposure to the x-ray and the local application of radium. None of these measures is adequate, and very few cases of actual cures of this lesion have been reported.

It is not within the scope of this presentation to enter into the anatomy, histology or pathology of these lesions. It suffices to remind you, in order that we may understand each other's terminology, that the tracheobronchial tree consists of the trachea dividing into its two main branches—the right and left main stem bronchi. The carcinomas which develop in these structures are either adenocarcinoma, arising from the numerous mucous glands which are distributed widely throughout the submucosa, or squamous cell, arising from the lining epithelium which is stratified and columnar.

For purposes of future reference, I call your attention to the fact that this tumor, as well as the adenomatous variety, is medullary in type, being composed chiefly of epithelial elements and remarkably free from dense connective tissue. This would seem to be a favorable factor in so far as radio sensitivity of these lesions is concerned.

Early in their development these tumors, as seen in Fig. 2, are usually small, cauliflower-like growths involving either the trachea or one or the other of the main stem bronchi, and it is of tumors of this stage of development, size and position that I speak.

Concerning the therapeutic possibilities of treating such tumors, I cannot enter into the controversy concerning the use of x-ray

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Fig. 1. The trachea dividing into its right and left main stem branches.

therapy or diathermy. Experience would indicate that these are, to say the least, inadequate.

Since the early tumors involve the upper portions of the bronchus and trachea, successful surgery is technically impossible. The site of these tumors which ordinarily makes surgery impossible, is, fortunately, the chief factor favoring the application of adequate doses of radium.

There appears upon Fig. 3 a white circle enclosing the lower portion of the trachea and the upper segment of both main stem

Fig. 2. Early carcinoma, right main stem bronchus viewed from behind.

bronchi. It is within the enclosure of this circle that the majority of tracheobronchial malignant neoplasms find their site of origin. This site is particularly favorable for radium therapy, because, as may readily be observed, there exists in most instances a sufficient segment of bronchus below the lesion, and a sufficient length of trachea above it to permit radiation beyond the limits of visible tumor. Submucosal extension, within the lymphatics or in the epithelium itself, can therefore be brought to fall within the zone of adequate radiation. This principle of establishing an effective field of radium activity above, below and to each side of the tumor I believe to be of the utmost importance.

In overcoming obstacles of adequate radium application to tumors of the bronchus lies the sole value of this method which I am about to propose. Its limit of value, of course, is the theoretical limit of value of radium emanations in the treatment of cancer elsewhere in the body.

Fig. 3. The site of origin of most tracheobronchial tumors is located within the white circle.

The method, in brief, consists of filling long, hollow tubes with ordinary radium needles containing the salt and implanting these tubes so that they fill the entire length of the main stem bronchus and portions of the trachea. These tubes are designed to fit the needs of individual cases and a great number of varieties are possible. By this means the entire bronchus and lower segment of the trachea is exposed to whatever dosage of radium is deemed necessary. This, therefore, includes the tumor and healthy tissue above, below and

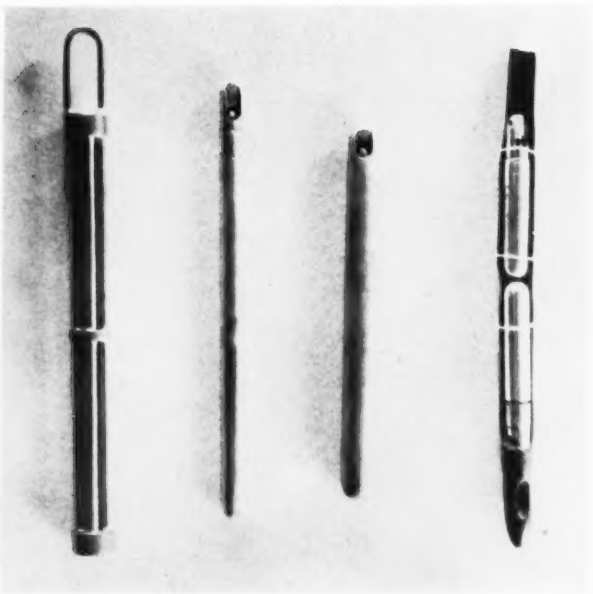

Fig. 4. Various containers for intertracheobronchial radium therapy.

around it. Radium is continuously in situ for as long a period of time as is necessary to give the required dosage.

The longer tubes, as shown in Fig. 4, are 10 centimeters, or 4 inches, in length and from 6 to 9 millimeters in diameter. A determination of the size and type tube necessary is made at a preliminary bronchoscopy. Features to be considered are the length of the tumor, its position in the tracheobronchial tree, the side involved, the contemplated dosage of radium and, as I shall shortly dwell upon, the degree of preliminary pneumothorax which has been instituted.

The principle of tubes Nos. 2 and 3 is simply that of a hollow aluminum container with a screw cap. They are approximately 10 centimeters long and 7 millimeters in diameter. Radium "needles" in varying numbers are inserted into their lumen in tandem. The tubes themselves are of platinum of either one-half or one and one-half mm. thickness. This tube is primarily for the right main stem bronchus, and when inserted occupies a position such as is shown in Fig. 5, filling the bronchus from its lowermost depths to well above the bifurcation.

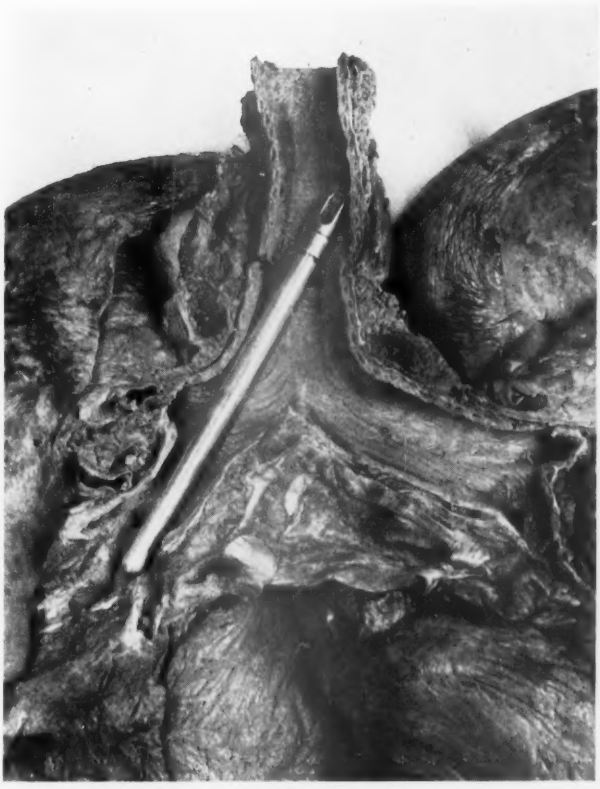

Fig. 5. A solid aluminum container in situ in the tracheobronchial tree.

Under certain circumstances it is necessary that during the period of installation air be allowed to pass freely into the lung. This is absolutely impossible with the solid type contained shown in Fig. 5. Accordingly, there has been designed by one of us (C. E.) a tube which has the general appearance of an elongated bird cage. It consists of a series of hollow struts supported by thick circular bands of metal, one in the center and another removable band or collar at each end. The radium needles are placed in the lumen of the hollow struts and retained in position with the screw collar. The loop at the end is for the purpose of bronchoscopic removal of the tube. This is a very efficient apparatus, inasmuch as its bulk is sufficiently great so that it is almost impossible to displace it by the most

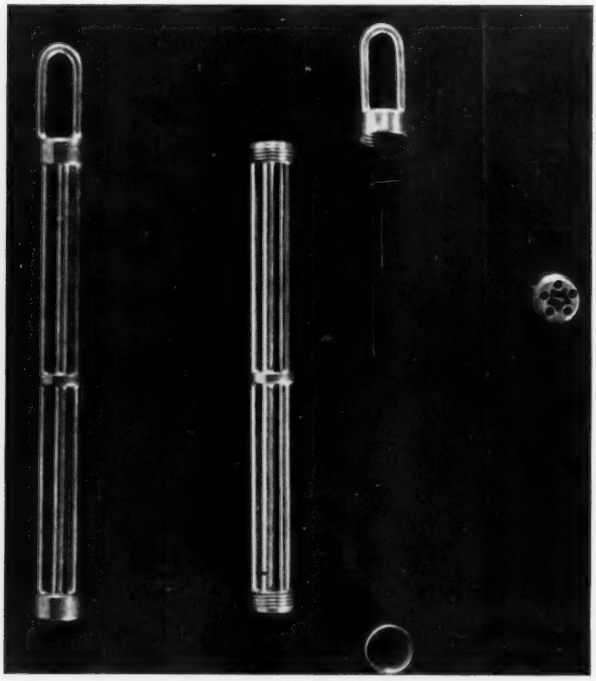

Fig. 6. The "Bird-cage" container. In the central figure the collar on each end has been unscrewed. At the right is an end-on view with the collars removed. Radium needles are inserted into the struts through the five small holes. Both ends are alike.

severe paroxysm of coughing. It also permits close approximation of the radium to the bronchial wall and an even distribution of radium emanations throughout the length and circumference of the bronchus. When conditions warrant, the use of this tube is my method of choice.

Fig. 7 demonstrates its appearance lying in situ in the tracheobronchial tree. The circle surrounding the site of early carcinoma is superimposed, so that we may see how long a segment of tracheobronchial tree above and below the lesion is irradiated. The importance of this cannot be stressed too often.

We come now to an entirely different type of applicator. This is shown lying in situ in the right main stem bronchus. The distal

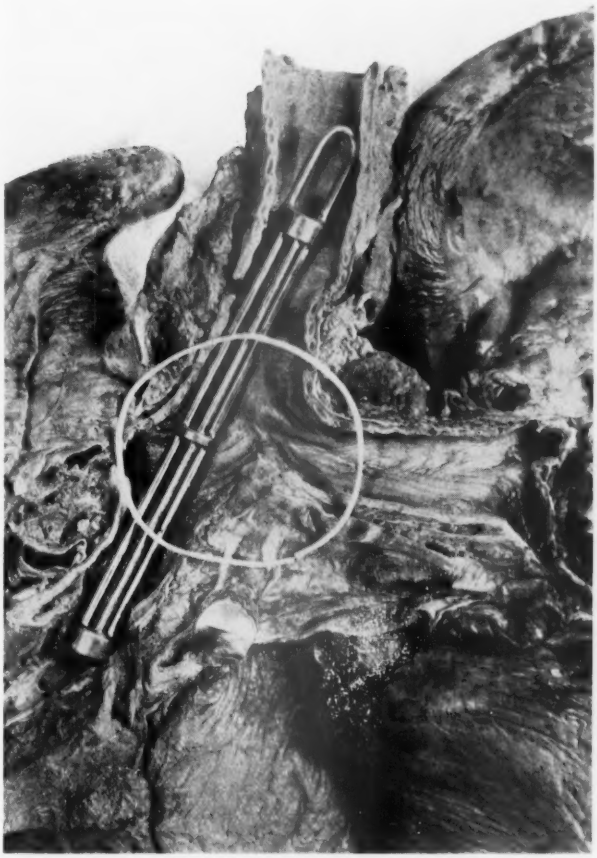

Fig. 7. The "Bird-cage" container lying within the tracheobronchial tree. The site of most early carcinoma is demonstrated by the white circle.

portion of a rubber catheter is used as an outer jacket. For purposes of demonstration half the diameter of the catheter has been removed, but of course this is not true when the catheter is in actual use. Within the catheter are placed two, or even three, very short aluminum containers. This produces the effect of a segmented tube with great pliability. It is especially useful in the left main stem bronchus where the relatively greater acute angle at which the bronchus comes off the trachea makes the use of a rigid tube difficult.

Fig. 8. Short aluminum capsule-like containers lying in tandem within a catheter. Note that the butt ends face each other. Otherwise the cap of the lower container would represent an area of non-radiation.

The question of radium dosage is best left to the radio therapeutist. This will vary in each case, depending upon the extent of the area over which the radium is to be distributed. But in general we have tried to deliver a dosage of from 4,000 radium r to 6,000 radium r to the tissue at a depth of 3 to 4 millimeters from the applicator. The time necessary to deliver this amount of radiation will vary with the size and content of the applicators, but usually extends over a period from 30 to 65 hours of continuous treatment.

It is obvious that the insertion of so large a foreign body into the bronchus for so long a period of time must of necessity cause obstruction to the inflow of air with resultant massive collapse of the lung. This may be overcome in one of three ways:

1. By maintaining the tube in situ for relatively shorter periods of time and repeating the insertion in order to make up the total dosage;

2. By using hollow tubes of which the container nicknamed the "bird cage" is the best example, but even this tube, after 15 to 20 hours, may become occluded by thick mucopus or blood.

It does, however, represent a considerable improvement in this respect over the original solid container.

The third method of preventing massive collapse is our present method of choice. At the suggestion of Dr. Jacob Abowitz, preliminary pneumothorax was instituted. This procedure wrote a new chapter in the development of the technic. The pneumothorax is made as complete as possible. We later found it advantageous to maintain this for many weeks following the radium insertion. It presents such outstanding advantages that its use has become routine except for those cases where adhesive bands prevent collapse from taking place. In those instances, where it seems propitious, I deem it worth while to have the bands severed with the thoracoscope and pneumothorax then instituted.

The advantages of complete preliminary pneumothorax are many. The lung having been previously compressed, massive collapse cannot possibly occur either during the period of treatment or during the period of radium reaction, which comes from two to six weeks later. The collapse of the lung diminishes the possibility of hemorrhage. If the compression of the lung be sufficiently great, it is reasonable to assume that a greater area of lung tissue is brought into the field of effective radiation. The lung is put completely at rest so that movement of the container is less apt to occur. Obviously if no air enters the lung, no effective cough can be produced from that side and the radium container is less apt to be coughed up. For these reasons we heartily endorse the use of preliminary pneumothorax as an integral and vital part of this method of treatment.

Fig. 9 demonstrates the combined use of complete pneumothorax in conjunction with the insertion of a large bird cage type of radium needle container. Under such circumstances the container can remain in situ almost indefinitely. Note the outline of the completely collapsed lung, the pneumothorax and the radium container.

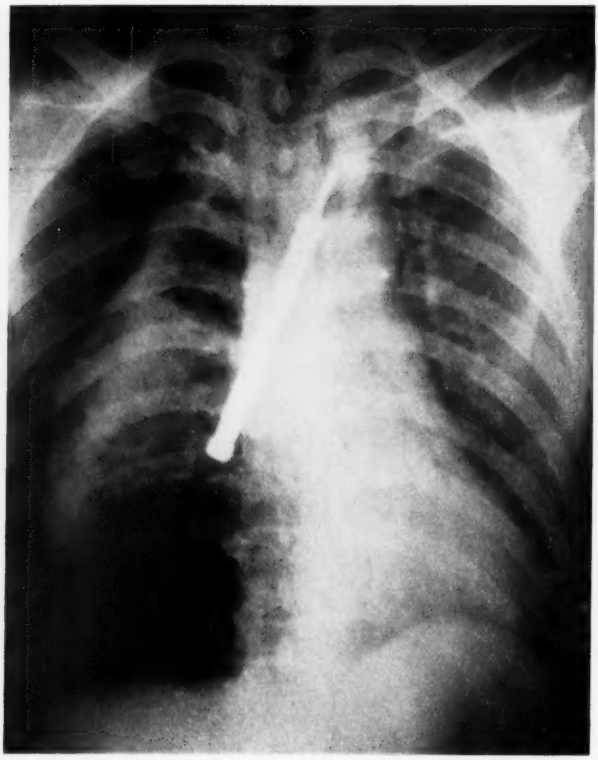

Fig. 9. The "Bird-cage" container in situ in combination with pneumothorax.

The pre-operative preparation is of some importance. The patient is admitted to the hospital the day previous. That night two capsules of nembutal or amytal are administered for sleep. On the following morning prior to the operation, the patient receives 1,000 to 1,500 cc. glucose intravenously. An additional large dose of nembutal is given and an hour later ¼ grain morphin and 1/150 grain of scopolamine. These latter two are repeated just prior to insertion, if it seems necessary. I prefer to have the patient brought to the operating room in semi-coma.

The anesthetic is local as for any bronchoscopy, except that 1 cc. of 10 per cent cocain is dropped into the involved bronchus.

Technic: First the bronchial lumen is re-established by removing as much of the tumor as is necessary with either a punch forceps or fulguration. The technic of actual insertion varies according to the size of the capsule and the side involved. Some containers are difficult to remove, and if this be anticipated a long woven silk string is firmly tied to the proximal end, prior to the insertion. If the capsule is of sufficiently small diameter to fit through a bronchoscope, this is inserted and the container grasped with a foreign body forceps. It is then introduced into the bronchus through the bronchoscope and under direct vision carried as deeply into it as possible. This method is especially indicated in left sided lesions. If the container is too large to fit through the bronchoscope, as is the case with the "bird cage," it is inserted through a direct laryngoscope. The larynx is exposed. The container, seized in the grasp of a laryngeal alligator action forceps, is inserted between the cords. It is then carried down blindly until it has entered the bronchus, usually the right. The bronchoscope is then inserted and a bronchoscopic suction cannula or foreign body forceps used to push the container deep into the bronchus and its position checked by bronchoscopic vision. The string, if one has been used, is allowed to project from the mouth or nose and attached to the side of the face with adhesive tape. The string, however, tends to provoke cough, but since it is a safety factor is probably well utilized. In some cases we use it; in others, we do not deem it necessary.

The patient is immediately taken to x-ray and the position of the tube checked fluoroscopically. If the patient can be made to cooperate it is well to have him cough in order to determine whether or not a coughing paroxysm tends to displace the tube. He is then placed in bed in a sitting position with sideboards for protection. During the period of treatment the patient is kept narcotized with frequent large doses of morphin. Fluids and caloric intake are provoked by intravenous glucose. If coughing is profuse, which is not very often the case, provided morphin be given in adequate doses, or if it seems that copious amounts of mucus or pus are present, the tube is removed to be reinserted again after a day or two of rest. Divided doses are possible, but usually the tube can be maintained in situ for the entire calculated period. Occasionally the patient coughs up the smaller tubes, in which case it may block the larynx, so that vigilance on the part of the nurse is necessary. If this occurs she can easily remove the container by traction on the string, which is always used with tubes of small diameter. Intentional removal, if no string has been provided, is done through the bronchoscope with foreign body forceps. The postoperative course ordinarily is smooth

and the patient remains quietly at home, or is only slightly active for a period of about six weeks.

The pneumothorax is maintained by repeated installations of air as often as necessary. During this period, coughing and expectoration are apt to be increased. Before the end of six weeks a radium reaction is to be anticipated. This occurs in the form of edema of the bronchus, which may be so extensive as to completely occlude it, or nearly so. The advantages of having maintained a pneumothorax are now apparent, since it prevents massive collapse of the lung, reduces the likelihood of lung sepsis and hemorrhage during the radium reaction. The temperature may be slightly elevated, the cough becomes slightly more profuse and is productive of small or moderate amounts of yellowish gray sputum, sometimes foul. A certain amount of general debility occurs. This reaction is apt to be of several days' to a few weeks' duration, after which it gradually subsides. The pneumothorax is then allowed to absorb and the lung re-expands.

A word about complications: The obvious possible complications are hemorrhage, lung sepsis, pulmonary massive collapse and postoperative stricture of the bronchus. In about twelve cases we have had one massive collapse during treatment, but this occurred prior to the use of pneumothorax. One fatal hemorrhage developed eight weeks postoperatively. We have had no case of severe pulmonary sepsis and no case of stricture of the bronchus sufficiently severe to require dilation.

Concerning results following this method of treatment, I can say very little. It has been proven to our satisfaction that radium in adequate dosage administered according to this technic, in properly selected early cases, can result in complete local disappearance of carcinomata of the bronchi. We feel, for the following reasons, that when radium is indicated, this is the method of choice: (1) The technic is simple and relatively safe; (2) It provides adequate, carefully measured radium dosage; (3) Radiation of equal intensity throughout all portions of the tumor, and (4) Radiation of healthy tissue above, below and around it.

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XXX

BENIGN STRICTURES OF THE ESOPHAGUS: NEW METHOD OF GRADUAL DILATATION*

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Most benign strictures of the esophagus follow the swallowing of corrosive solutions which have been taken by accident or with suicidal intent. These substances frequently produce death within a few days from necrosis of the mouth, pharynx, esophagus or stomach. If the patient survives the immediate effects, the tissues of these organs undergo swelling, edema, desquamation and finally distortion from contraction of the scar tissue which is formed. Although this contracture is gradual, the final closure to the passage of food and fluids is apt to be a surprise, indeed often a shock, to the patient and frequently to his physician. In this emergency, which usually arises in the third week, there is an intense desire on the part of the afflicted to secure immediate relief. This relief too frequently takes the form of a gastrostomy and the opportunity to secure a more permanent and satisfactory solution of the difficulty is postponed or even lost because of the lapse of time which permits the stricture to become more complete. Gastrostomy should be regarded as an admission on the part of the surgeon that he is unable to give relief otherwise and must employ this side-tracking procedure to keep the patient alive. It should be remembered, however, that any ordinary individual will live between thirty and forty days without food, provided ample fluids are given. Our modern methods of the administration of intravenous glucose in normal salt solution in quantities up to 3,000 or 4,000 cc. daily are so common after major operations that it is hardly necessary to argue that a gastrostomy can be delayed until all feasible methods of penetrating the stricture have been tried and found to be unsuccessful.

The management of such a case should include, first, hospitalization to gain complete control of the patient and to facilitate the administration of intravenous fluids in ample quantities; second, reassurance of the patient to allay his fears regarding the possibility of

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starvation, and to forestall the demand for an early gastrostomy which is so frequently put forward by those surgeons whose experience offers no other solution; third, penetration of the stricture for the preservation of a passage, however small, at the earliest possible moment. It should also be remembered that a hungry patient is infinitely more cooperative in the carrying out of procedures which require penetration and dilatation of the esophagus than one with a full stomach, and that this early period should be utilized in an attempt to accomplish something of a permanent nature.

Once the patient has been gotten under control, how can the esophageal stricture be penetrated? Three methods are available: First, by the passage of instruments over a silk thread or fish line; second, by the use of an esophagoscope; third, by a method which I wish to propose which involves the use of a Levine duodenal tube. The choice of these methods will vary according to circumstances, but without doubt the safest of all is the passage of dilating instruments over a silk thread, provided it is possible for the patient to swallow it, as the instrument will follow the thread, whatever direction it may take. The second method, the use of the esophagoscope, is excellent if used by an expert, but is equally dangerous in the hands of the untrained. It has the distinct advantage of an actual view of the esophagus, but unfortunately only the upper end of the stricture can usually be seen and it gives little information about conditions below this point. If instruments are passed by this method the visible stricture may be successfully penetrated, but if there are others lower down they must be dealt with more or less blindly.

The method which I have to suggest, the passage of a Levine duodenal tube, like the others, must be carried out with care and judgment, remembering that injury or perforation of the inflamed and friable esophagus is always a real danger. The lumen of the ordinary Levine duodenal tube ends at the eye. The tip is soft and relatively uncontrollable. To overcome this objection and render the tube more rigid it is advisable to use a stylet. This stylet may be purchased with the tube or it can be made from a piece of tonsil wire. For the purpose of making the tip rigid a hole is burned with a hot needle or probe from the eye of the tube down into the tip, making it possible to extend the stylet into this portion of the tube. The tube and stylet are grasped near the base with a hemostat and the tip is then introduced through the mouth into the esophagus. It is not introduced through the nose because it is usually too inflexible to make the turn on the posterior pharyngeal wall. As the tube passes down the esophagus the stricture is located. If it does not pass by gentle pressure 4 or 5 cc. of liquid petrolatum are injected into the lumen

of the tube with a hypodermic needle below the point where it is grasped with a hemostat so that it will emerge at the point of the stricture for lubrication. Gentle pressure is then made, slowly rotating the tube, so that the tip will exert pressure in the various directions. Frequently the tube will slide through the stricture into the stomach and the emergency which would otherwise call for gastrostomy has been averted, as this provides a satisfactory method of feeding the patient. If this method proves unsuccessful and the patient has been able to swallow a thread, the hole previously burned into the tip of the Levine tube can be extended completely through it so that this short canal in the tip can be threaded over the guide line, allowing the tube to be passed into the stomach. Even if the esophagoscope is used and the passage is opened with a bougie or dilator, it is always wise to follow it immediately with a Levine tube for the purpose of feeding the patient and to maintain the lumen at all times. Once the Levine tube has been introduced, whatever method may have been employed to introduce it, it should never be removed except for the purpose of *immediately* substituting another of greater size. Although it is usually necessary to pass a tube containing a stylet through the mouth, once it has entered the stomach the stylet may be withdrawn and the free end of the tube brought out through the nose where it is fastened in position with adhesive tape. This is easily done by introducing a catheter through the nose until it can be seen in the pharynx where it is grasped, drawn out and attached to the Levine tube by means of a glass connecting tube. The spliced tube is then brought out through the nose. Successive tubes usually do not require the use of a stylet and can be passed directly through the nose.

The customary method of dilating these strictures with bougies or dilators, either with or without anesthesia, has never been satisfactory, because any interval dilatation produces so much trauma by sudden stretching that hemorrhage and edema follow and predispose to recurring contraction and stricture. The use of the method proposed in this discussion is based upon three principles, supported by the following facts: First, gradual stretching of any stricture produces less reaction and is more permanent than any type of sudden dilatation. This has been our experience in dilating the urethra, ureter, rectum and other passages of the body. Second, any sinus through soft tissues tends to enlarge from the continuous presence of a rubber tube which passes through it. This is well known in the use of a rubber tube placed in an empyema or gastrostomy opening or in an ordinary abdominal drainage wound. Although the tube may fit tightly at first, in a very few days the opening will increase in size to such an extent that leakage will occur around it or the tube will

become loose enough to slip out. This type of dilatation is so gradual that reaction of the tissue is minimal. Third, the impact of food as the result of swallowing is an important factor in producing and keeping up the secondary inflammation which leads to final closure of the esophagus. This is evidenced by the fact that when food is administered through a gastrostomy opening as a substitute for feeding by mouth, the esophageal constriction will frequently reopen and again allow food to pass. This occurs in obstruction caused by carcinoma as well as that caused by other types of stricture.

In the light of these facts, it seemed reasonable that the use of *a series of Levine tubes of increasing size* would act in the same way in the esophagus and would produce a gradual and more permanent dilatation of the strictures as well as provide a means of feeding the patient during the process. In my experience it has been a very successful method of treating these strictures, as the following typical cases will illustrate.

REPORT OF CASES

CASE 1.—Mr. M., forty years of age, suddenly became unable to swallow even liquids, twenty-one days after drinking three ounces of lye. I saw him thirty-six hours later. He was dehydrated and hungry, and it was evident that a complete closure of the esophagus had occurred. Either early relief of the stricture or a gastrostomy was necessary. The patient was very co-operative. A No. 12 French Levine tube was prepared with a wire stylet, as described above, and introduced through the mouth. The stricture was encountered near the cardia. Five cubic centimeters of liquid petrolatum were injected through the tube to lubricate the point of stricture. Gentle pressure was then made, slowly rotating the tube by swinging the free end from side to side. After a few trials the tip slipped through into the stomach and the stylet was withdrawn. To get the base of the Levine tube through the nostril proved to be a simple matter. A small rubber tube was inserted into the nose, fished out through the mouth, and attached by a glass connecting tube to the Levine tube, and then drawn back through the nose. From this time on the patient was fed through this tube which was left in place continuously, except when another was substituted for it. By the fourth day the stricture had enlarged enough so that the tube could be moved in and out easily and the patient could swallow fluids around it. It was then removed and the next size Levine tube (No. 14) was substituted. It was not necessary to use a stylet to introduce it. Three days later it was replaced by a No. 16 French tube, and then a No. 18 after another three days. Then it was discovered that Levine tubes of a larger size were not made.

I found, however, that by cutting off the tip of an ordinary catheter and the bell end of another, the two catheters could be spliced together over a short glass connecting tube and would produce a substitute for a Levine tube which was of the proper length to reach from the nostril to the stomach. As catheters come in all sizes, a graduated series of "Levine tubes" could easily be made. Though no tube larger than size No. 22 French will ordinarily pass through the nose, the fact that this tube was made of two parts and joined in the center proved to be a blessing

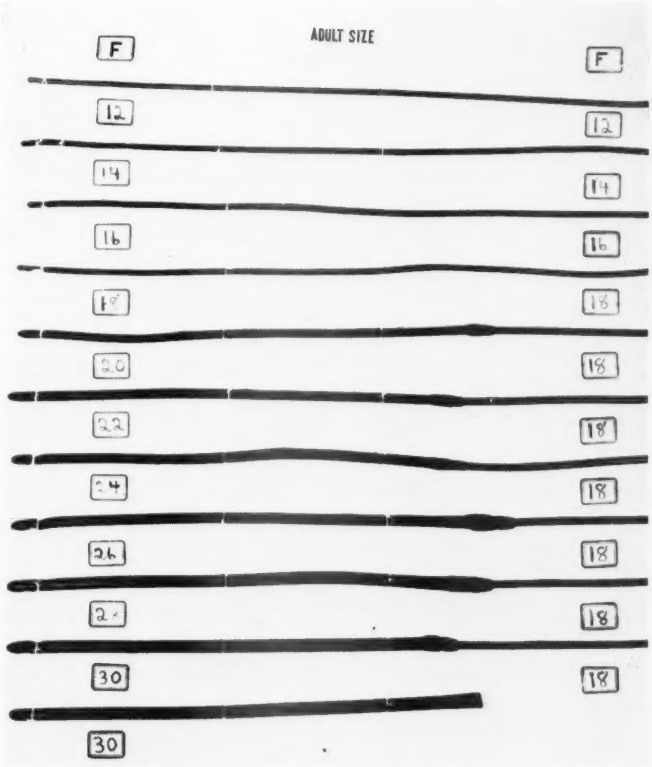

Fig. 1. Adult size. "Levine" duodenal tubes, sizes 12 to 18 F. Composite "Levine" tubes, sizes 20 to 30 F. Length of tube in esophagus is calculated for the individual patient so that the joint will remain in the pharynx. Connections are made by means of short glass or copper tubing. The catheter illustrated last is made about 10 cm. longer than the esophagus. A thread is attached for anchoring it over the ear and for its withdrawal. This tube is used to maintain dilatation, at first between meals and later only during the patient's sleeping hours. Instead of a composite tube the two pieces can be vulcanized together or made as one tube. In this event the small end must be drawn through the posterior nares each time it is changed.

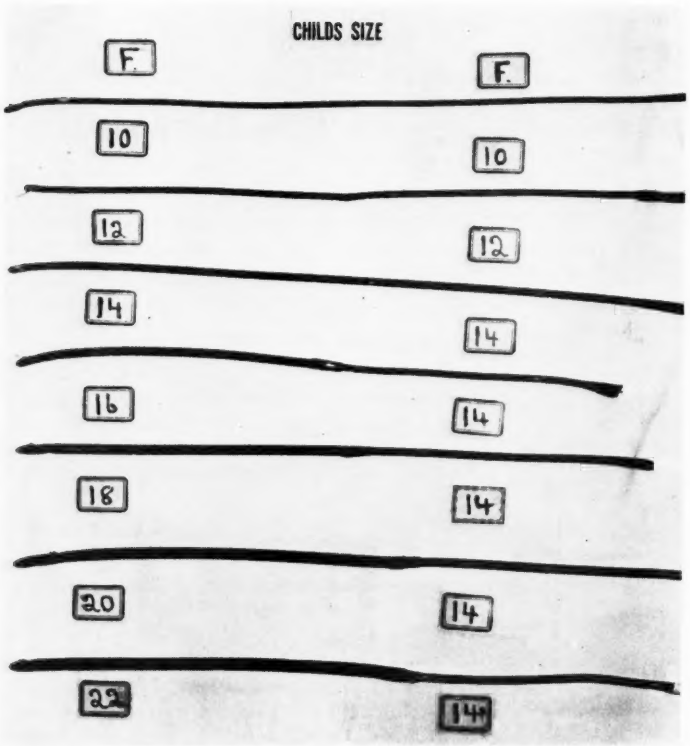

Fig. 2. Child size (for age 22 months). Length of esophagus about 15 cm. With children too young to co-operate all tubes are introduced through the nose, the larger sizes after shrinking the nose with adrenalin. The joints should be sandpapered until accurate and smooth.

in disguise and made it possible to attach a larger tube to one small enough to pass through the nose and thus to continue the gradual dilatation and feeding in the same manner. Whenever the tube became loose and freely movable in the esophagus the lower part was fished out through the mouth, the next larger size was attached to the small tube remaining in the nose and the large tube was reinserted into the esophagus, thus gradually dilating the stricture.

At the end of twenty-four days the esophagus had been dilated sufficiently to allow the passage of a No. 30 French catheter. During this entire period the patient had been fed through the tube, not only for the purpose of convenience but to prevent secondary inflammation of the esophagus. This point is of importance, if dilatation is to be secured in a minimal time. When dilatation had been completed the tube was removed and the patient was allowed to eat his meals in the

normal way. Contracture, however, is always to be feared. As a precautionary measure the tube was replaced between meals for the purpose of maintaining the dilatation. For this purpose I found that it was more convenient to have the patient use an ordinary No. 30 French catheter to which a thread had been attached, for anchoring it over his ear. He soon learned to insert this tube himself after each meal and withdraw it by the thread at the next meal time.

Thirty-three days after the dilatation was begun the patient went back to his regular work, able to take care of himself. Because contracture has been so prevalent by our previous methods it seemed wise still to maintain the dilatation, and the patient was instructed to adopt the routine of leaving the catheter in the esophagus at night (that is, approximately eight out of twenty-four hours), removing it during the day. As a matter of fact, on retiring he would remove his false teeth and toupe and swallow his tube, while in the morning he repossessed his artificial devices, removed the tube and hung it on the wall for the day. He followed this regime for one year, after which the esophagus had returned to a state so nearly normal that all restrictions were removed and there was no tendency to recurrence.

Case 2.—J. N., aged twenty-two months, was admitted to the Children's Hospital on June 23, 1936, with a stricture of the esophagus of about two months' duration. The esophagus had closed about three weeks after ingestion of lye. Bougies had been passed regularly about once a week and whenever the child became unable to take sufficient food.

The child was underweight, weak and anemic, and was able to take small quantities of pureed foods only with great difficulty. On June 24, esophagoscopy and visualization with thin barium before the screen showed a stricture one inch or more long at the level of the aortic arch. From that date to July 17 bougies (from 8 to 16 F.) were passed under ether anesthesia at intervals of from five to seven days, to prevent starvation. At the end of this period the specialist in charge reported that gastrostomy seemed inevitable as it was impossible to maintain dilatation.

I saw the patient first on July 20, 1936. A number 10 F. nasal catheter was passed into the stomach and all feedings thereafter were given through the tube. The child received homogenized foods, milk and beef juice, malt tonic and twelve grains of ferrous sulphate daily. Gradual dilatation was obtained by means of larger catheters in series, until by August 20 (a period of 31 days), dilatation was sufficient to accommodate a number 20 F. catheter. The interval required for dilatation by each catheter varied from five to ten days. The criteria of sufficient dilatation to permit the use of the next size were (1) looseness of the tube, and (2) the child's ability to swallow fluids around it. The child gained weight and strength and the hemoglobin increased from 51 to 85 per cent. By September 8, 1936, the nasal tube was removed and feedings by mouth were resumed. The tube was reintroduced each evening and left until morning.

He continued to gain until he had another flare-up of his tonsillitis, which made reintroduction of the tube seem inadvisable because of the additional irritation. The entire procedure was then abandoned for a time, hoping that contracture of the esophagus would not occur. However, after about four weeks the patient again had difficulty in swallowing and the tube had to be again introduced with the aid of an esophagoscope under anesthesia. Again the patient did very well, gained weight and was apparently upon the road to a good recovery until the epidemic of influenza occurred. He contracted this disease, developed a double lobar pneu-

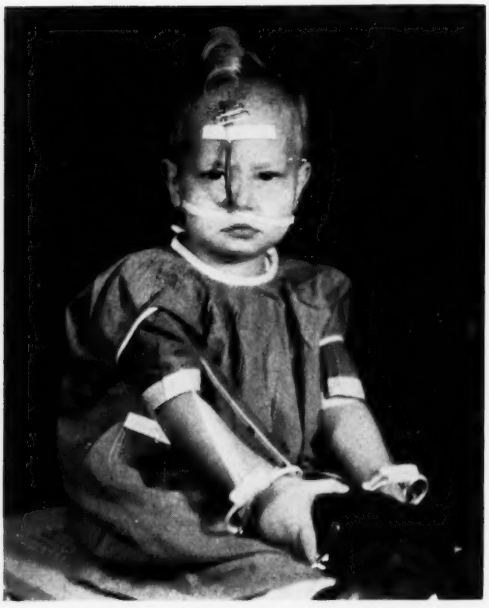

Fig. 3. J. N., aged 22 months. Tube in place fixed by adhesive to lip and cheeks and to forehead. Paper clip or small bulldog clamp used to close tube. Elbow restraining device in place to prevent child from removing tube. (Usually necessary only for first few days.)

monia, which resulted in his death. I am presenting for your inspection the esophagus of this child, which will give you some idea of the thickness of the scar tissue which was formed and the magnitude and duration of any procedure which would have to be carried out to secure a permanent dilatation. Considering the age of the patient, his extensive tonsillar, sinus and ear infections and the length of the esophageal stricture, it is not probable that a more difficult case will present itself for some time.

These two case reports illustrate a method of treating strictures of the esophagus which is simple, comparatively safe and, I believe, much more permanent than most other methods employed. The primary introduction of a Levine tube with a wire stylet is not without danger of perforation if not intelligently used. Bougies, dilators and the esophagoscope, however, require equal care in their use or they, too, may perforate or injure the esophagus.

In both cases described the strictures were extensive and severe. Mild cases may be dilated in less time, but even greater precautions

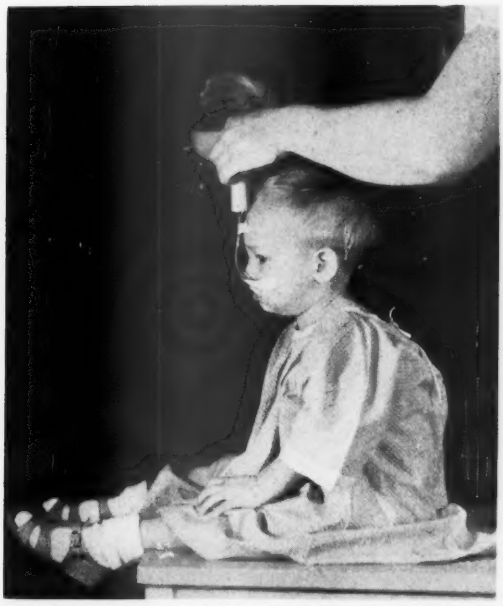

Fig. 4. J. N., aged 22 months, being fed by gravity through the barrel of a syringe. A triumph or pressure syringe may be necessary for thick or pureed foods. Tube should be irrigated with water after each feeding.

must be taken to prevent contracture if the process is hurried. I have also tried stretching these strictures in the old way and then maintaining that dilatation by the use of the larger size tubes just as has been done following the more gradual method. So far the number of cases is too small to warrant any conclusions, but it would appear that the danger of injury or rupture of the esophagus and subsequent infection of the mediastinum is considerably greater. Moreover the ultimate success of either method may frequently depend upon the ability of the patient to pass the tube down his own esophagus for a long period of time, and a course of hospital training is necessary to secure his confidence and cooperation and to impress upon him the necessity of following this definite regime.

Someone has raised the question as to the disposal of saliva after the introduction of the Levine tube. This tube seldom fits so tightly that saliva will not pass around it, and if collections of saliva become

a factor at all it usually ceases within twenty-four hours after a change of tubes. As a matter of fact these tubes are not really dilators. They simply take up the slack in the opening as the tissues recede. It is only a principal applied to the esophagus which has been used in the urethra for years.

It has been suggested that this method might be useful in treating cardiospasm. It seems reasonable that it would be effective, as we at present employ dilatation, but I have had no experience with it up to this time.

Too much emphasis cannot be placed upon the necessity of anticipating the possibility of stricture of the esophagus after the ingestion of corrosive substances, if we are to prevent this occurrence. Either a silk thread should be introduced before stricture occurs, or a small Levine tube should be inserted through the nostril into the stomach as a precautionary measure. The thread can be anchored to a tooth and is less bothersome and conspicuous. The tube, however, not only maintains the passageway but permits food to be introduced in adequate amounts and without irritation of the inflamed area. From a practical standpoint, the thread can well be used first, to be followed by the tube if necessary.

I am well aware of the generally accepted teaching that no device should be introduced during the acute period and that frequently gastrostomy should be done early to put the esophagus at complete rest. While this dictum may have saved an occasional life, it is also responsible for a considerable number of patients destined to live with permanent gastrostomies and with the prospects of a limited longevity. Early gastrostomy allows the patient to take the management of the situation into his own hands and often lulls the attending surgeon into such a state of lethargy that the opportunity of a successful penetration is lost. A definite difference exists between penetration for preservation of the esophageal lumen on one hand and instrumentation for dilatation of the actual or potential stricture on the other. Desquamation is usually over or subsiding by the tenth to fourteenth day. The swelling is receding and scar tissue is forming but has not yet begun to contract. It is in this period that the thread or small Levine tube should and can be introduced with safety. Unfortunately the patient is getting better, is still able to swallow and does not wish to be bothered by either a thread or a tube. However, the alert and experienced physician must insist on these preventive measures if the morbidity of this condition is to be reduced or avoided.

In my opinion the following rule would be of great value: Every patient with a corrosive burn of the esophagus severe enough to

justify an early gastrostomy should have a prophylactic penetration of the potential stricture, and every patient developing a stricture of the esophagus following the action of corrosive agents should be allowed every reasonable means of its penetration before being subjected to a gastrostomy.

SUMMARY

1. Early introduction of a thread or Levine tube is necessary if impassable strictures are to be avoided.

2. The different methods of penetrating early strictures of the esophagus are reviewed and an original method employing a Levine tube is described.

3. A new method of gradual dilatation of strictures of the esophagus by the use of a series of Levine tubes of increasing size gives a more permanent and satisfactory result and affords a means of feeding the patient without gastrostomy.

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XXXI

ANATOMY OF THE BRONCHIAL TREE AND ITS CLINICAL APPLICATION*

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The marked advances in recent years in the diagnosis by radiology, and the treatment by surgery of lung diseases, has stimulated the study of the anatomy of the bronchial tree.

After the trachea has divided into the two primary bronchi, the secondary bronchi to the upper, middle and lower lobes have, of course, been recognized from their elementary constancy. When the tertiary bronchi are under consideration their apparent inconstancy has caused anatomists to fall back on the terms "dorsal," "lateral" and "ventral," which are frequent in textbooks on vertebrate comparative anatomy.

In some of the vertebrates, for example, the pig, the tertiary bronchi of the lower lobe can be seen coming off from the dorsal, lateral, ventral and mesial aspects in regularly diminishing size, from before backward, or cephalad to caudal. The stem apparently branches monopodially to the final main terminal bifurcation.

The terms "dorsal," "lateral" and "ventral" were used in human anatomy to describe the location of the lower lobe bronchi, whose orifices appeared in corresponding surfaces of the bronchial stem: the terms were meaningless as regards ultimate distribution and carried no salient memorizing data. Their apparent inconstancy prevented precision in description, and their further distribution was lost seemingly in a maze. As an example, Dwight Davis,[1] after describing the secondary bronchi according to their possessing two, or three branches, finished a paper dealing meticulously with over eighty specimens, by stating, "Since the arrangement of the bronchi is so

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varied it is difficult to reach a definite conclusion, except that each lung is different. No other organ, so far as I am aware, has such a varied arrangement of its fundamental structures." He used radiograms of lungs into which opaque mediums had been forced.

Kramer and Glass,[2] in 1932, published what we consider to be a most important paper. They showed that tertiary bronchi or those that come off the main bronchi are distributed with a constancy of 85 per cent, and that each tertiary bronchus is distributed to a special segment of the lung, forming what they term a broncho-pulmonary segment, whose limits can be delineated on the periphery, and whose relations to the chest wall can be defined. Kramer and Glass made use of liquid dyes in fresh inflated autopsy lungs. Nelson,[3] in 1934, published an article in which he corroborated the findings of Kramer and Glass.

We show here a simple diagram with their essential findings and another of their diagrams of the bronchial tree as modified by us, showing the distribution of the bronchopulmonary segments. They found that the constant bronchi of the main stem of the bronchial tree are the upper lobe, middle lobe, and, of the lower lobe, the apical, the mesial (or cardiac or infracardiac, or azygos, which is found only on the right side), the anterolateral and the terminal paravertebral and posterolateral.

It will be noted that their terminology conveys a topographic description that allows a ready visualization of the architecture of the lungs.

The anatomy of Kramer and Glass can be readily correlated with the rudimentary bronchial buds in the lungs of a nine millimeter embryo.

With the exception of the mesial, all bronchopulmonary segments have surfaces in contact with the chest wall, and are therefore accessible to surgery. The mesial or cardiac is so constant and accessible that surgical requirements can be met by a suction tube.

Kramer and Glass make a most important point that lung pathology is primarily confined to one bronchopulmonary segment. In lung abscesses it makes its way towards the periphery, setting up an adhesive pleurisy that effects adhesion to the chest wall. With a knowledge of bronchopulmonary anatomy, the surgeon can go through the adhesions safely, enter a lung abscess and avoid the disaster of entering the pleural cavity, or, what is worse, of going through normal lung tissue.

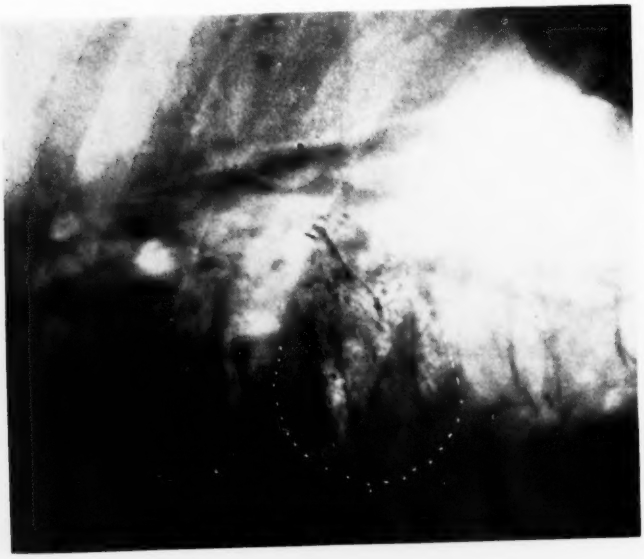

Fig. 2. Single filling, right subapical bronchus. Lateral view.

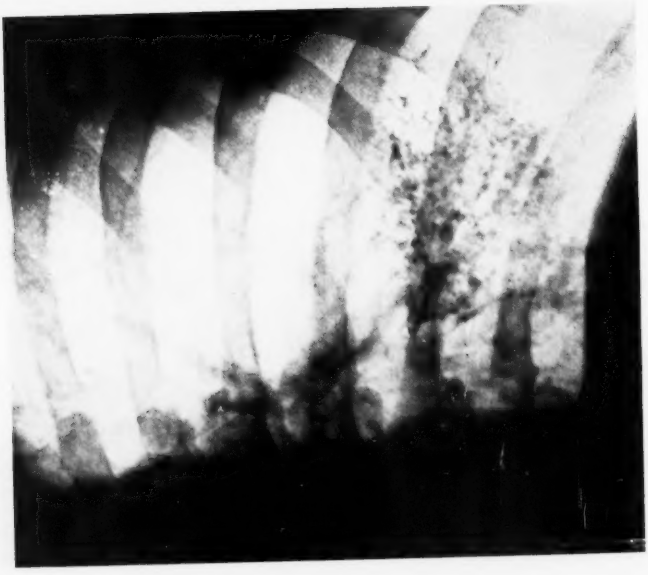

Fig. 1. Single filling, right subapical bronchus. P. A. view.

In our investigations into bronchial anatomy, we made use of fusible metal in autopsy specimens of human and other mammalia, the dissection and inflation of autopsy specimens, the examination of the lung in vivo by bronchoscopy and the instillation of lipiodol.

We used Wood's fusible metal, which consists of tin 1, cadmium 1, lead 2, bismuth 4. Thrice that quantity in ounces is a useful amount for a large adult human lung. This compound will melt in boiling water, but as it quickly cools and will produce only stumpy casts at that temperature, we found this method useless. In making casts of human lungs after the components are fused, the heating should be continued for at least another minute over an ordinary Bunsen burner. This will give a temperature of approximately 300 F. In small mammals the extra heating may cause the pleura to be perforated. In these cases, when the metal is fused, and the dross cleared, the resulting temperature of about 250 F. is satisfactory. The thorax of the specimen should be unopened, and the funnel used in the trachea should have a metal tube down within the spout, to allow the gases to escape from the trachea and avert spluttering of the metal, which is poured in a steady stream. After a few minutes the lungs are removed, and as much lung tissue as possible removed from the lung surface, without interfering with the terminations of the cast. Surface incisions are made into the lung, avoiding the bronchial branches. The specimen is then immersed in a 25 per cent solution of caustic soda for forty-eight hours. With toothed forceps the tissues can be removed by pulling them off in the axes of the local bronchi. The specimen will give an intimate knowledge of the anatomic points, and by holding them in appropriate positions whilst viewing specimens and radiograms one may clarify what may have seemed a hopeless problem.

Dissection and inflation can be carried out in fresh autopsy specimens. They can be put in 5 per cent formalin for twenty-four hours, then drained trachea downwards or suctioned. Wrapping specimens in the following preserving fluid will keep them soft and pliable: Two hundred grammes of arsenate of soda are boiled in three liters of water and added to seven liters of tap water in which a kilogram of acetate of soda has been dissolved. Three liters of glycerine are then added.

For dissection of the bronchi, blunt pointed curved scissors will give access to the plane of tissue round the bronchi. By teasing and finger dissection, all the branches mentioned can be readily displayed. They should be exposed for about half an inch.

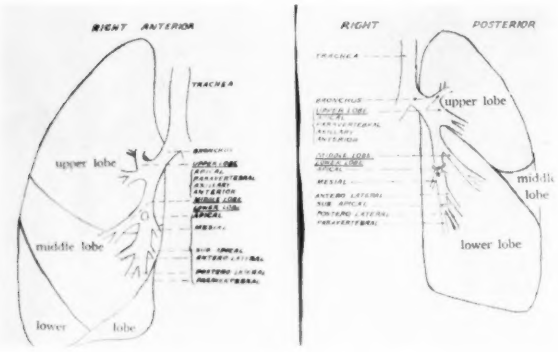

Fig. 3

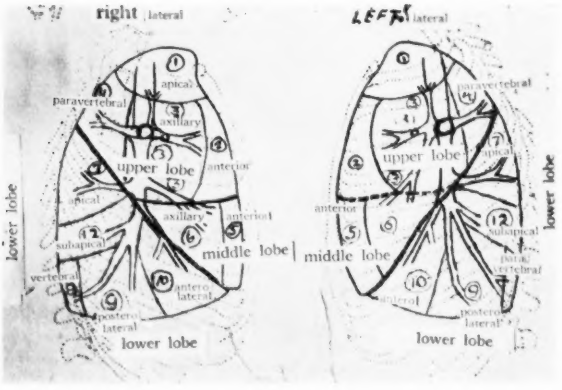

Fig. 4

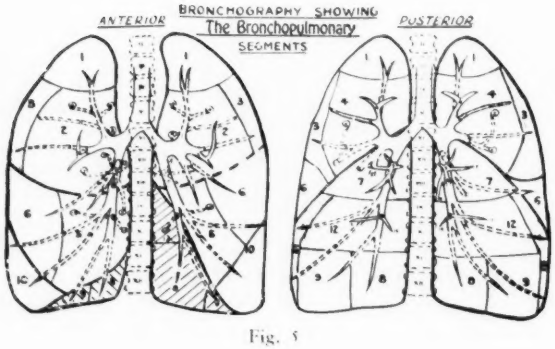

Fig. 5

If the main bronchi are incised prior to this, the specimen may be spoiled, as orientation may be lost. We have found that by the use of a Higginson's syringe and a suitable nozzle, inflation through the bronchus of each pulmonary segment will show on the surface its projection with clear demarcation. All apparent exceptions, in autopsy specimens, to our findings should be inflated for investigation.

The use of lipiodol has certainly advanced the study of bronchography. Unfortunately, the method generally used is to instill amounts varying from 5 to 15 ccs. down the trachea, or into a bronchus, and take radiograms. The result is a superimposition of bronchial shadows that may show gross pathology, but the separate bronchopulmonary segments can rarely be definitely determined.

We have adopted the method of single filling of the separate bronchial branches, after they have been cleared of discharge by suction, restricting the amount to 1 cc. for each. Separate fillings are valuable in the interpretation of nonfilled segments when the opaque lipiodol is obviously confined to one segment. The patient is then sent immediately to the radiologic department, where posterior to anterior and lateral views are taken. The slides will show how necessary views in two planes are for orientation.

We will now mention the major results of our investigations and will pass by the upper lobe, merely stressing the fact of the constancy of its four orifices and segments, and pointing out that the paravertebral bronchopulmonary segment, which is the first to come off from the dorsal aspect of the vestibule, in much larger than hitherto recorded. It is also, by the way, a site of predilection of tubercle invasion. The middle lobe is also a constant with its two branches. We have been concentrating our investigations on the variations of the lower lobe bronchi. The apical of the lower lobe is a constant. Indeed, it has been frequently shown that it is often a separate or partially separate lobe. The mesial or infracardiac is also a constant. The anterolateral is also a constant, but may have two openings—the anterior above and the lateral below. The bronchial tree ends by dividing into two terminal bronchi, the inner or mesial paravertebral and outer or lateral posterolateral. Our principal finding is that in varying positions between the apical above and the terminal branching below are found one or two bronchi that have escaped recorded notice by all anatomists, surgeons and pathologists to whose writings we have had access.

It is necessary to go to comparative anatomy to get an explanation of their occurrence. In the cat we get a bronchial tree that has anatomic constants that are unequivocally homologous with the bronchi of the bronchopulmonary segments detailed above in the human.

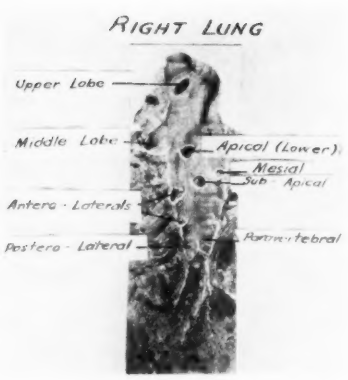

Fig. 6

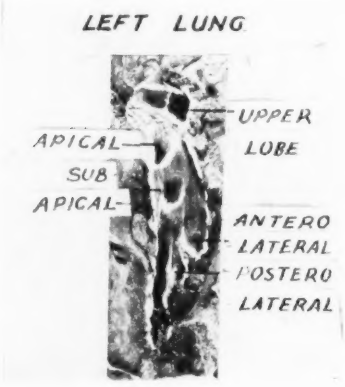

Fig. 7

Fig. 8

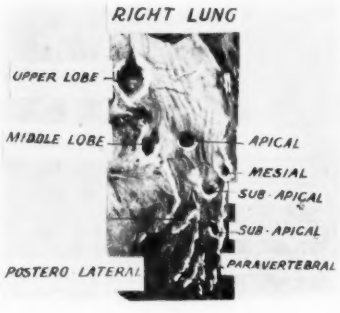

Fig. 9

From and including the apical of the lower lobe, there is a series of sprouts or branches in descending magnitude down to the tips of the terminal bronchial branches. They are mainly on the dorsal and lateral surfaces, except on the terminal bronchi, where they are also seen on the ventral surface, and one or two on the mesial. Between the apical and the final division of the stem into the paravertebral and posterolateral, there is on the dorsal aspect a well marked sprout on both right and left stems. We have termed it, in a previous communication,[1] the posterior intermediate.

Unfortunately, this term is used in describing one of the air sacs, apart from the lungs proper, that occur in birds. The term subapical has been adopted as the better term.

Homologues of this may be seen in the mammalia, and we have had the opportunity of examining and corroborating this in the following: the monkey, sheep, pig, deer, kangaroo and dog.

We will now show a series of metal casts of some mammalian lungs. In these the dorsal branches of the lower main stem bronchi are seen in regular sequence. Beneath or caudal to the first or apical, the homologue of the human subapical is constantly seen above the final bifurcation into paravertebral and posterolateral. Its distribution to the area between the apical above and the paravertebral below is constant. In the human left lung we have found it situated on the mesial aspect, or the dorsal, or at times lower down on the dorsal, just at the orifice of the paravertebral. Twice clinically we have found it on the lateral and twice on the ventral. This is in accord with our findings in some of the mammalia as previously mentioned. However, on the right side we have found it constantly either just below the apical on the dorsal surface, mesially below the mesial, and on the dorsal aspect, just above the final bifurcation of the stem. We have proved by means of metal casts, inflation, lipiodol and x-rays, its distribution to the territory between the apical above and the paravertebral below, and naturally have found it to be the subject of pathology. In the latest published contributions to the anatomy and pathology of the lower lobe, it has been incorporated in either the apical or paravertebral segments. We suggest that with knowledge of the sites of the subapical bronchi, there are no obstacles in the way of the description of the lung in terms of bronchopulmonary segments with the surety of ordinary anatomic textbooks.

With more intimate anatomic knowledge, clinical work will be more concise, and further investigations can be made into the causation of predilection for certain sites in such diseases as tuberculosis, sili-

Fig. 10. Metal cast of dog's lung.

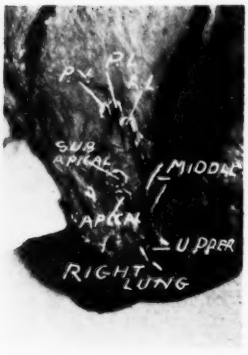

Fig. 11

Fig. 12. Showing areas delineated on infiltration of Fig. 3.

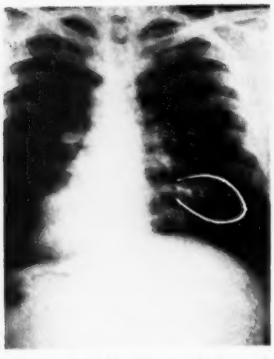

Fig. 13. Single filing, right subapical bronchus. P. A. view, alternative to large.

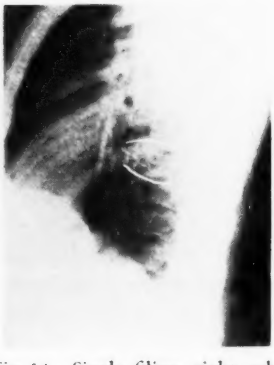

Fig. 14. Single filing, right subapical bronchus. Lateral view, alternative to large.

cosis, or miners' disease, and lung abscesses, can be investigated. Dr. Gwynne of Auckland, who has been with us from the inception of these studies, is publishing, with the speaker, in the Medical Journal of Australia, an article that will give data on topography that will be of interest to radiologists.

The infra-cardiac, or azygos, or mesial, as we term it, is well marked as a separate lobe in our specimens of the monkey, cat, pig, duiker, sheep, dog, and kangaroo. In the human it is incorporated in the right lung.

Sir Colin MacKenzie, of the Australian Institute of Anatomy, at Canberra, has kindly sent us drawings of the wombat, in which it is well developed, and of the koala, or native bear, in which it is apparently absent. As is well known, in snakes and snake-like lizards the left lung is vestigial, the right lung being lengthened. Illustrations of the lungs of the Australian copperhead snake and of the Queensland death adder bring out these points.

We will now discuss the clinical application of bronchography.

As the other bronchi mentioned are constants, the orifices of the sub-apical are the variants that may give rise to nonrecognition or confusion. The identification of a bronchus is of great importance, as the orifice of a diseased segment generally shows indications of disease. Septic foci, such as abscesses and infected tubercular cavities, are frequently met with. Their cure or relief is greatly hampered by obstruction of the bronchi leading to them. Disease products and mucus frequently so clog the action of the all important cilia, which lift the mucus and dèbris upwards, that the cavity becomes practically closed, and the destructive anaerobic bacteria may increase the broken down area surrounding the cavity.

There is no known drug that can, in therapeutic doses, alter the activity of the cilia, and mucus cannot be either dissolved or absorbed in the body. Hence the importance of opening up or clearing the affected bronchi. In suction we have an important means of effecting this. In dealing with the infection of cavities the removal of débris must be our main objective. Antiseptics, although useful, must play a minor part, compared with the potent connective tissue cells or macrophages that line the air sacs and detach themselves from the walls to engulf foreign matter or débris. Antiseptic injections may be used. Solutions of an aqueous isotonic type are much more effective than oily ones and are apparently harmless. Experiments on dogs showed that isotonic solutions, with the equivalent of the salt

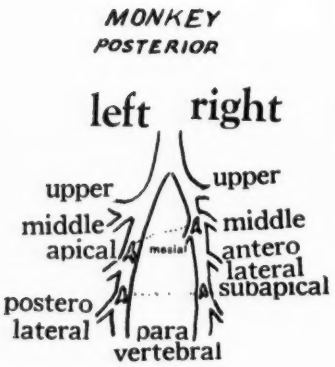

Fig. 15

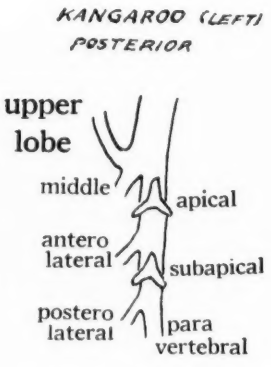

Fig. 16. Stump cast drawings.

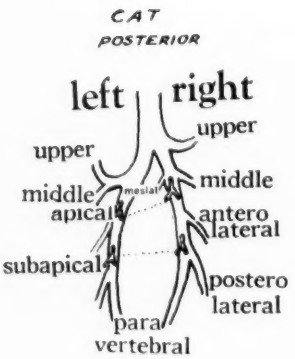

Fig. 17

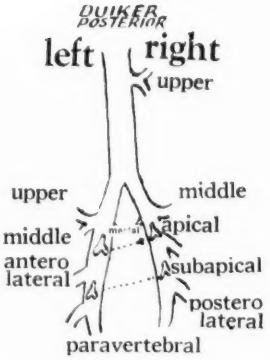

Fig. 18

content of the blood, can, by drop flow, be instilled into the bronchi to the amount of six liters, without apparently causing harmful changes in the general body condition or subsequent serious microscopical lesions in the lungs. Colored aqueous solutions were found to have passed through the finer bronchi into the air sacs and on to the rich lymphatic system. The fluid did not come back by the trachea. Use is now made of this fact to deal with tubercular and nontubercular cavities. Once the bronchi leading to them, and if possible the cavities themselves, are suctioned out, suitable antiseptics in aqueous solution can be introduced in amounts varying from 30 to 500 cc. A Murphy drip is used at the rate of one drop in two seconds. Some of the more potent antiseptics that have been made in the last few years are now available. We have made use of "metaphen" in the strength of 1-10,000. It is an organic mercury compound that in laboratory tests will, in a dilution of 1 in 60,000, in the presence of serum, destroy the streptococcus in five minutes.[5]

It is thus one of the most potent destroyers of the streptococcus hemolyticus, which is the most commonly present pus producing organism in lung disease. In therapeutic doses it is apparently innocuous to the kidneys. Bronchoclysis is the name given to this procedure by Mandelbaum.[6]

He introduced a catheter into the lower part of the bronchial stem. We are showing by cinema that we can introduce the catheter by means of the bronchoscope into the bronchus of the exact segment involved. The radiograms subsequently show a cloudiness of the lung tissue that disappears within a week, when the procedure may be repeated. Lower respiratory diseases bulk largely in mortality statistics, and contribute largely to disabilities entailing serious economic inefficiency. We are grateful to the pioneers for their work in this special branch of medicine, but trust that others will add to their structures, and help to minimize the incidence of respiratory diseases, in which chronicity is an ever-increasing menace to relief.

Mr. W. A. Fairclough, F. R. A. C. S., has followed this work and, being a keen natural historian, has taken a great interest in the comparative anatomy. The cinema film which he has made himself is an index of his kind co-operation.

We wish to express our indebtedness to Mr. J. A. Sandford, who made the line drawings for us. He thinks that he is repaid by having had a staple removed that had been thirty-six years in his lung. The x-ray staff of the Auckland Hospital have given invaluable assistance, as have the staffs of the Pathologic Laboratory and the medical superintendent's office.

64 SYMONDS ST.

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XXXII

RELAPSING ALTERNATING PERIPHERAL FACIAL PARALYSIS*

PIERRE VIOLE, M.D.

LOS ANGELES

Oppenheim[1] states that "facial palsy is the most common neural paralysis." One sided facial palsy is a rather frequent occurrence, and recurrent attacks are not unusual. Alternating recurrent attacks, on the other hand, are comparatively uncommon, if one is to be guided by the scarcity of cases of this type reported. The synchronous involvement of both sides by peripheral palsy is exceedingly rare, while congenital, traumatic, inflammatory or toxic facial diplegias of the central type have been frequently encountered in perusing the literature. There is no way of determining the relative frequency of unilateral or bilateral facial palsy. In 1833, Sir Charles Bell[2] quoted a communication from a colleague who reported, in French, the bilateral involvement of the facial nerve of luetic origin. According to this report, a girl, sixteen years of age, had a paralysis involving the left side of the face, which was followed eight days afterwards with a similar paralysis involving the other side. Under anti-luetic treatment complete return of function ensued. Recurring or relapsing form of Bell's palsy is of much greater frequency than is commonly supposed. In an extensive survey of 200 cases Remak[3] stated the incidence to be 3 per cent, while, according to Bernhardt's[4] extensive statistical study, the percentage was as high as 7.2 per cent. Merwarth,[5] reviewing 192 cases, estimated the incidence to be 7.7 per cent. This type of facial palsy has been previously reported by Huet and Lejonne,[6] Orbinson,[7] Merwarth[5] and others. Merwarth has reported the greatest and most varied number of cases.

As the title of this paper indicates, we are particularly interested in the alternating, recurrent or relapsing type of facial palsy. By this is meant the clinical entity in which is found a peculiar tendency to recurrences or multiple attacks of facial palsy. In the relapsing type

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From the Department of Otolaryngology, School of Medicine, University of Southern California.

the same side of the face is subject to recurrences while in the alternating type the two sides are affected at different times. For the latter, according to Hunt,[8] the term "relapsing alternating" is the more exact or specific. The interval between attacks may vary greatly as in the original report of Bell,[2] in which eight days elapsed between attacks, while in the other cases reported the lapse of time has been months or years. The etiologic factors to which can be attributed the causation of the relapsing alternating type are the same as those encountered in the more frequent clinical types of peripheral facial palsies.

In the so-called "a frigore" type there is a tendency to definite local reactions to temperature changes or infection which is evidenced by a facial palsy. An analogous situation occurs, which is commonly observed in the association of tonsillitis and sore throat with lumbago, sciatica and other rheumatic manifestations, with their proneness to recurrences. Perineuritis of the facial nerve similar to that existing in brachial and sciatic perineuritis of rheumatic origin is a theory advanced by many as being the pathogenesis of rheumatic or refrigeration palsies. Dehiscences or anomalous course of the nerve would favor such lesions. Any swelling within or about the nerve in the fallopian acqueduct would be immediately evidenced as pressure palsy. According to Despaigne,[9] the nerve would naturally be more susceptible, due to the pressure of a congenitally narrowed stylomastoid foramen are fallopian canal. This theory would explain some of the familial or hereditary types which are occasionally encountered, the constitutional tendency or diathesis being also transmitted. It is interesting to note that this tendency need not be limited to one member of the family, as Oppenheim[10] has reported repeated attacks in three members of a family all of whom were diabetics. The familial tendency was also emphasized by Neumann[11] who, in 1887, reported family histories in thirty-seven cases of facial palsy.

Other etiologic factors that should be considered are the presence of acute or chronic otitis media and mastoiditis with disease of the adjacent bone or of the nerves themselves. Infections such as lues, tuberculosis, acute epidemic encephalitis and instances of multiple neuritis must also be considered. Blood dyscrasias as well as some of the metabolic diseases may be precipitating causes. Drugs, such as arsenic, mercury and salvarsan, etc., may be considered as irritants, besides toxic factors such as typhoid, influenza, diphtheria, acute contagious diseases and bacteremias, as well as those commonly known as "foci of infection." Otitis media, syphilis and diabetes were found to be present in 33 per cent of the cases reported by Bernhardt.[1]

The symptomatology of this disturbance is identical with the more common affliction which is limited to one attack, with the exception that the patient is doubly concerned and becomes rather apprehensive as to future attacks. This, I believe, exists to such a point that there might be said to be a premonition or intuition as to the oncoming attack.

Pain, as well as disturbance of the sense of taste, are probably the most significant symptoms as regards the ultimate outcome of the involvement. These two symptoms have been greatly stressed by Tumarkin[12] in his recent survey of this subject. Pain varies considerably in severity in the relapsing type of palsy. It is usually located in the ear, mastoid or occipital region and frequently radiates to the trigeminal distribution. According to Hunt,[13] Webber[14] and Testaz,[15] pain is accounted for through the sensory function of the facial nerve. It is also a well established fact that the fifth nerve plays a definite role in the pain phenomena which exist with disturbances of the facial nerve. Typical migraine has existed in some instances, as has been reported by Rossolimo.[16]

Disturbance of taste is a very common accompanying symptom whenever the lesion is situated at or proximal to branching of the chorda tympani. This is shown by a loss of the sense of taste in the anterior two-thirds of the tongue on the affected side. Oftentimes this is very difficult to determine, due to the rapid spread of the testing agent and the profuse collateral nerve supply in the tongue. Many patients state they have a mineral or metallic taste in the mouth when the distribution of the chorda tympani has been disturbed.

Whenever the lesion is located proximal to the stapedius branch the synergistic action of the stapedius muscle and that of the tensor tympani must of necessity be unbalanced. This may be evidenced by hyperacusia or dysacusia. Even vertigo and other vestibular phenomena may be experienced.

When accompanied by herpes zoster (Hunt's syndrome) herpetic blebs or scars may be seen upon the tympanic membrane, external auditory canal or concha. Hyperesthesia of this same area may exist.

It is maintained, and I believe justly so, that if a thorough and complete otologic examination and study had been made of cases of peripheral facial palsy at the onset or just preceding the onset, that definite changes would have been observed in the middle ear.

REPORT OF CASES

CASE 1.—Relapsing, alternating facial palsy; four attacks.

History.—G. L., male, aged 40, salesman by occupation, whose family history is negative as regards facial paralysis. The patient had a facial palsy of sudden onset which occurred November 15, 1932. When he consulted me four months later there was a typical peripheral facial nerve palsy, involving the right forehead, eye and face. The sense of taste was not impaired, nor was pain noticed. There had been no history of ear involvement. Examination revealed no hearing impairment, the septum was deviated to the right, and the tonsils were infected. The patient was advised of the tonsils as possible foci of infection. During the course of a month the paralysis showed distinct improvement with complete recovery within six months. The second attack occurred on August 28, 1934, practically two years after the initial attack. In this episode the paralysis involved the left side. As in the case of the first attack there was no accompanying pain, nor was the sense of taste impaired. This paralysis was also discovered upon awakening. The paralysis was complete. Examination of the ear, nose and throat revealed findings as above with the addition of a dark right antrum. The right antrum was found to be infected and was treated. The paralysis improved, and the face returned to normal function during the course of treatment. Again the patient was advised that the tonsils were the probable focus of infection. He was next seen on December 28, 1934, at the office and stated that he believed a recurrence of paralysis was imminent. He complained of pain in the region of the right mastoid and was experiencing gradual lack of control in the upper lip. The hearing was not impaired, nor was the sense of taste. There was persistent nasal mucoid discharge. These symptoms had been present four or five days prior to the consultation. Upon examination wrinkles appeared more flaccid on the right side of the face than on the left. The otolaryngologic findings were similar to those of the previous examinations, with the persistent darkened right antrum. At this point the patient consented to a submucous resection and tonsillectomy. Upon arrival at the hospital on the morning scheduled for the operation I found the patient afflicted with a distinct right sided facial paralysis. Submucous resection and tonsillectomy were performed December 31, 1934. Usual postoperative care was given and the paralysis disappeared within a month. Again on March 8, 1935 the patient reported to the office stating that on February 21, 1935, he had felt the suggestion of the onset of another left sided paralysis. This one, however, was of very short duration and involved only the closure of the left eye. Up to the present date there has been no recurrence of paralysis.

General physical and neurologic examinations were repeatedly made. No abnormal findings other than reported above were found. Blood Wassermann and the other usual laboratory tests were found to be negative.

Present Status.—Both sides of the face at the present date have from all appearances returned to normal without any evident residual involvement.

Comment.—This, I believe, is a typical example of relapsing alternating facial palsy; two of the attacks were on the right side, and one and the suggestion of a second occurred on the left. It was quite difficult to convince the patient of the relationship between the facial paralysis and a focus of infection. From the foregoing it is quite natural to presume that the nerve was involved in the fallopian canal distal

to the chorda tympani, as no gustatory or hearing impairment was experienced by the patient. Hyperesthesia of the concha or evidence of herpetic scars or vesicles was not noticed. Pain was not a prominent symptom, but it is interesting to note that after the second attack the patient had a premonition of the coming disaster. Also it was of interest to note the distinct relationship of the elimination of foci of infection to the course of recovery.

CASE 2.—Alternating facial palsy.

History.—Miss R. G., female, aged 40. Case was observed at the Los Angeles General Hospital, May 15, 1929. The patient felt perfectly well when suddenly and without warning she developed an intense pain in the right ear. This occurred after being exposed to cold night air. This lasted throughout the night and the following morning disappeared. She noticed, however, that the hearing in the right ear had become impaired, as well as the sense of taste. In the afternoon, twenty-four hours after the onset of the earache, she discovered that the right side of her face was completely paralyzed. The impairment of hearing soon improved, but the facial paralysis persisted for a period of two months, and then began to respond to physio-therapeutic measures, such as massage, diathermy, quartz lights and galvanic stimulation. Six months after the onset of the disturbance normal function apparently returned.

On March 17, 1931, the patient developed an upper respiratory infection which persisted for one month. Shortly afterward she developed a severe earache in the left side. This was unaccompanied by other signs or symptoms of acute otitis media and lasted for two weeks, at the end of which time the pain disappeared, but she developed a complete left facial paralysis with impairment of hearing and disturbance of taste. The onset of this attack was attributed by the patient to having been exposed to a draft while driving. Mastoid films were negative as well as the laboratory examinations, including urinalysis, blood studies and repeated Wassermann tests. Associated with the development of this second paralysis was a moderately severe pain, which radiated over the entire left side of her head. It was several weeks before this pain subsided. In spite of considerable physiotherapy the improvement was very slow, and to date complete return of function has not occurred.

Present Status.—At the present time the patient is undergoing no treatment of any description. There is still some residual involvement of the left facial muscles which affects the closure of her left eye, with tearing aggravated upon mastication. She is still unable to wrinkle her forehead, and what is more annoying and creating more discomfort is the fact that food still becomes lodged between the teeth and cheek. This condition is accentuated by a distinct impairment in swallowing.

Comment.—It is interesting to note that in this case pain with involvement of hearing was experienced, as well as disturbance of taste. Extreme lacrimation, which is more pronounced upon mastication, still persists, especially on the left side. This is a typical example of synkinesis, which Dorland defines as an unintentional movement accompanying a volitional movement. We are justified in assuming that the pathologic process has affected the nerve proximal

to the nerve supply of the stapedius muscle or in the vicinity of the geniculate ganglion. Tumarkin, who has greatly stressed the occurrence of pain and the disturbance of taste as clues to prognosis in given cases of facial paralysis, explains the synkinesis by assuming that the fibers intended for the salivary glands wander into the lacrimal. The salivary fibers, of course, travel along the chorda tympani normally, but if some get lost and enter the greater superficial petrosal nerve they will eventually locate in the lacrimal gland by traversing the vidian nerve, the sphenopalatine ganglion, the sphenopalatine nerve, the superior maxillary division of the fifth and the temporal nerve which communicates with the lacrimal nerve. This is evidence of a lesion involving the facial nerve at the geniculate ganglion. Naturally lesions located here or proximal to the ganglion are not amenable to relief by decompression of the nerve, as advocated by Duel. Tumarkin is of the opinion that in all probability the group of 20 to 25 per cent of the cases of Bell's palsy that do not recover belong in this category. The functional disturbance of the digastric and stylohyoid branches of the facial nerve is clearly demonstrated in this patient by her deglutitive impairment. Vertigo and hyperacusis, which are doubtless due to the involvement of the stapedius branch causing an imbalance of the synergistic action of the stapedius and tensor muscles are of less significance.

SUMMARY

My observations in these cases lead me to agree with Tumarkin that the presence of pain at the onset of a facial palsy and the disturbance of taste are of great prognostic significance. In the first instance the patient did not complain of either of these symptoms and recovery apparently is complete. These two symptoms were definitely present in the second case reported, and there is still noticeable residual involvement. The phenomenon of synkenesis, as described by Tumarkin, plainly exists in the second patient. Disturbance of the facial nerve also undoubtedly influences the act of deglutition as is demonstrated in the latter case.

The premonition experienced by the first patient, I believe, is of interest and probably has existed in other instances.

These two cases of relapsing alternating peripheral facial paralysis are presented mainly because similar cases must be of more frequent occurrence than is evident by the scarcity of reported instances in the literature. Is it possible that they are not considered of sufficient interest or importance to be more frequently mentioned?

1930 Wilshire Blvd.

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XXXIII

CONGENITAL CHOANAL ATRESIA: TWO CASES OF COMPLETE BILATERAL OBSTRUCTION*

BENTON N. COLVER, M.D.

GLENDALE, CAL.

UNILATERAL ATRESIA

Unilateral choanal atresia obviously is less serious than the bilateral obstruction and may remain unsuspected for years. It may be discovered in the routine examination of a long series of rhinologic patients in a public clinic. It may be first diagnosed when the parents of a child so afflicted seek to ascertain the reason for persistent nasal discharge and inability to clear one side of the nose by blowing. Such patients have often been seen previously by various physicians, and surgical operations on the nose and throat have been performed without relief of the symptoms and without establishment of the true diagnosis. These patients may complain of little discomfort or may be definitely distressed for years by the unascertained anatomic deformity. Thomasson reports the case of a man with atresia of one choana who at 29 years of age "had never discovered that he did not breathe as other individuals." In not a few cases in which the diagnosis is made, either the parent or the patient positively declines to have the necessary corrective surgery attempted.

Cavanaugh, in the course of discussion of Lebensohn's paper, reported the case of a 12-year-old child from whom the tonsils and adenoids had been removed, the latter on three different occasions; the inferior and middle turbinates had been removed and the attending physician was reported to have considered an ethmoid operation on the affected side of the nose. When the correct diagnosis of unilateral choanal atresia was made the mother declined to permit the necessary operation to be done. The author discovered two patients with unilateral atresia in the course of his practice, neither of which would consent to operation. These cases have not been reported.

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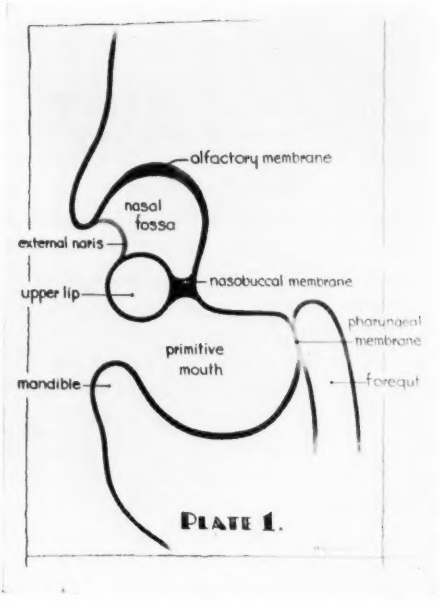
BILATERAL ATRESIA

Bilateral choanal atresia is a very grave occurrence. It has undoubtedly been the unsuspected cause of many deaths within the first two or three days of life. The rhinologist owes it to the obstetrician, to the pediatrician and to the general practitioner to emphasize its gravity properly and to make clear its role as a possible cause of the symptoms in cases of immediate difficulty in nursing or breathing, in cases of so-called asphyxia neonatorum and of "blue baby," and possibly even in some cases wrongly diagnosed as arrested respiration. The low incidence of the diagnosis in vital statistics tables or in medical literature is no measure of the true importance of the condition as a hazard to the newborn, but is in reality evidence of the condition having been overlooked. Asphyxia neonatorum may be due to the aspiration of amniotic fluid, meconium or mucus. It may also follow as a result of birth injuries or pressure to the head. Nevertheless, the dyspnea may be due to obstruction of the choanae, in which case it is evidenced when the baby normally closes its mouth. The importance of keeping this possibility in mind cannot be overemphasized. To know this fact and to act on it immediately and properly will conserve an infant life here and there that otherwise would be promptly snuffed out (Rogers).

HISTORY

The occurrence of congenital choanal deformity was first recorded in 1829 by Otto in Breslau. His observations were not made on the living and are rather unsatisfactory. He merely says, "In congenital closure of the hinder opening of the nostrils, the palatine bones are very much deformed." Apparently he had not had opportunity to observe examples of the more common choanal closures.

Not until 1853 does the first mention of this condition in a living patient appear in medical literature. The first surgical operation for the condition was performed in this first case of bilateral atresia ever reported. The operation was successfully performed by Emmert. The patient was a seven-year-old boy. If there had ever been previous clinical recognition of this obstruction, it is not established by recorded evidence. Voltolini reported cases in 1868 and 1870. Betts published in 1876 the report of a case diagnosed at or shortly after birth. J. Solis-Cohen pointed out a case in 1880 in which the prominent symptom was serious suffocation. In a series of autopsies, likewise reported in 1880, on infants dying with asphyxia neonatorum, Ronaldson declared this condition to be frequently the true cause of death. He failed to give details as to frequency.



Plates 1 to 4 are diagramatic sketches showing the progressive development of the nasal, oral and pharyngeal cavities, with the choanae. Arranged by Alfred Shryock, M.D., Dept. of Anatomy, College of Medical Evangelists.

Schroetter, in 1885, reviewed all the reports that were found in the library available to him. He mentions ten cases reported up to that date, quoting Otto, Emmert, Lushka, Voltolini, Bitot, Zaufal (two cases), Betts, Frankel and Sommer. He did not have available a report of the case of J. Solis-Cohen or the postmortem studies of Ronaldson. He also failed to note a second case reported by Voltolini as mentioned by Garretson. Schroetter adds the report of the case of a young woman of 19, in whom bilateral atresia was successfully relieved by him by surgical operation. He used a cautery, a burr and caustic potash; he later repeated the cauterization, then used a chisel and finally did several more cauterizations, all the work being carried over the course of many days. In 1886 Hubbell studied seventeen cases which he believed included all cases up to that date.

Through the intervening years a gradually mounting series of cases has appeared in the literature. By 1906 MacKenty quoted Taxier as bringing the list, not all verified, however, to 98. In two cases

noted by Taxier the infants died of asphyxia. Lebensohn, in reporting a new case in 1923, brings the authenticated cases to 170. He points to the statement of Richardson, in 1914, that the number of observed cases had increased greatly in the preceding twenty years, a tribute to the growing number of intelligent workers in rhinology and to the more widely diffused knowledge that such a deformity may occur.

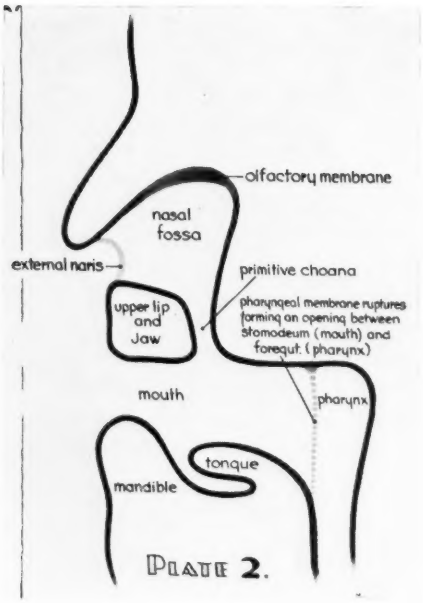
In 1927 Grove and Mahoney each stated that the total number of cases reported to that date "probably does not exceed 180." James and Hastings, in 1932, during the course of a discussion on nasal obstruction before the Section on Odontology of the Royal Society of Medicine, stated that "some 180 cases of congenital choanal atresia have been described and of these about a quarter are bilateral." It is indeed unfortunate that a clear statement of the type of obstruction is not made in all the cases that have been reported. McNaught states that atresia, either unilateral or bilateral, is, in his opinion, much more frequent in occurrence than has been generally supposed. Roth and Geiger and others concur in this belief.

INCIDENCE

Some observers state that the unilateral cases are more common, as did Clark, in 1898. It is quite possible that a considerable number of unilateral cases have been observed and correctly diagnosed but never reported, because the patients declined to have surgical operations for relief of the obstruction.

On the other hand, Richardson, in 1914, declared that the bilateral cases predominated in the proportion of three to one. Those who subscribe to this view may assume that the majority of cases of asphyxia neonatorum and some other cases of death within the first 72 hours after birth, are probably due to this malformation. Evidence is lacking and there is no sound foundation for the assumption. The only way that the facts may be determined is by routine examination of all newborn babies in a number of scattered obstetrical clinics, observing both in-patients and out-patients. Only when a great number of infants have been routinely examined may the frequency of incidence and the ratio between unilateral and bilateral atresias be intelligently established.

In the Ear, Nose and Throat Department of the Royal Infirmary of Edinburgh during a period of twenty years (1907-1926), 27,863 nasal patients were seen in the services of A. Logan Turner and J. S. Fraser, and among these patients there were six cases (0.02 per cent) of unilateral atresia and no cases of bilateral atresia. However, Stew-



art, in the article where these statistics are presented, reports two cases of bilateral congenital atresia in one family, occurring in his own practice. The patients were sisters, 18 and 15 years of age. Mention is made of a third child (male) in this family, who died within 48 hours after birth. He had attacks of dyspnea, was rather bluish and died of asphyxia. The possibility of this being a third case in the same family is strong. The attending physician was not clear as to the cause of death and thought it was due to "involvement of the lung."

Phelps feels that choanal atresia cannot be said to be hereditary, although there is on record one family in which the mother, her two sisters, two daughters and a son had this malformation. Evans reports the case of a child three weeks old who had choanal atresia. The parents of this child had four other children whose history strongly suggests the presence of the same deformity.

DEVELOPMENT

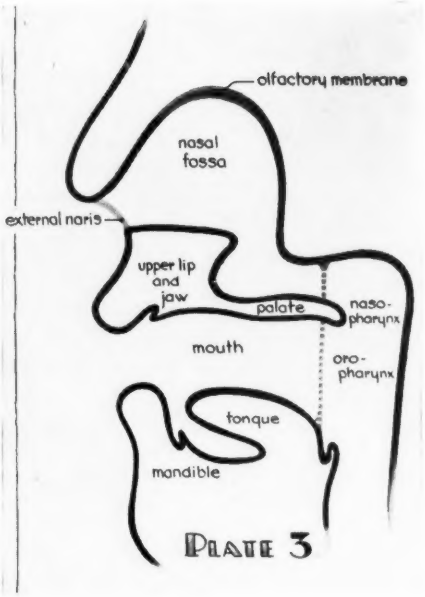
In the development of the nose the nasal chamber is at first a pit (olfactory fossa) and later becomes a blind pouch-like fossa

(nasal sac). In the early stages it is adjacent to the oral fossa. The epithelium of the nasal fossa soon comes into relation with the epithelium of the oral fossa. This apposition forms the nasobuccal membrane. Attenuation of this membrane to the point of rupture forms the primitive choanae. The choanae are separated from each other by the primitive nasal septum. In some cases there apparently is a certain amount of mesodermic tissue interposed between the epithelial layers of this nasobuccal membrane. Lack of rupture of the nasobuccal membrane or the formation of an epithelial plug which later becomes organized and thus blocks an already patent choana, may be observed in fetuses or in newborn babies.

Anterior to the primitive choanae lies the primitive palate. In the further development of the facial region the primitive palate is extended backward by the union of the palatal processes in the formation of the secondary palate. At the same time the primitive nasal septum extends backward and downward. This carries the primitive choanae backward until a communication is established with the cephalic termination of the pharynx (the nasopharynx). This connection determines the location of the definitive choanae. As a forerunner of this relation the membrane between the oral fossa (stomodeum) and the foregut, called the pharyngeal membrane, ruptures, bringing the mouth and the forming nasal chambers into direct relation with the pharynx. It is possible that a part of the pharyngeal membrane below the level of the forming palate may rupture while the portion above the palate, in relation to the nasal fossae, does not rupture.

The definitive choanae are bounded medially by the vomer (posterior border of the septum) and the nasal crest of the palatal bone; above by the alae of the vomer and the body of the sphenoid bone; laterally by the median plates of the pterygoid processes of the sphenoid bone and the perpendicular plate of the palatal bone; and below by the horizontal plate of the palatal bone. The muco-periosteum over this bony framework is continued backward from the nasal fossae into the pharynx (Bailey and Miller; Jordan and Kindred; Keith; Schaeffer).

It would appear from the developmental history and these various relationships that at least three structures might participate in the closure of the posterior choanae: first, the nasobuccal membrane; second, the bucco-pharyngeal membrane, and third, some part of the bony structure forming the funnel-like passage which constitutes the posterior choanae.



Richardson gives a classification of the atresias that apparently justifies such an assumption. He names the possibilities as follows:

1. Membranous. Such an obstruction is located posterior to the choanae in the nasopharynx, but in contact with the nasal orifices. This membrane extends upward from the soft palate at its junction with the hard palate. This is evidently the remains of the pharyngeal membrane.

2. Osseous. Such an obstruction is located about a millimeter anterior to the free border of the choanae. It thus presents a depression or dimple as palpated or viewed from the nasopharynx. This would arise from the nasobuccal membrane (the two epithelial layers with a film of mesodermic tissue between them).

3. Developmental. Such an obstruction is due to an overgrowth or malposition of the bones entering into the structure of the choanal canal.

Schaeffer states his belief that spontaneous rupture of the atresic mass, especially if thin and membranous, may occur in early infancy.

Garretson states that the bony obstruction may be as thick as 12 mm.

DIAGNOSIS

The diagnosis of complete bilateral choanal obstruction depends upon the age at which the patient is seen. The newborn suffers in a typical manner from dyspnea. The racial habit is to breathe through the nose with the mouth closed, and indeed even when the mouth is open (James and Hastings). When the infant attempts to do this, it is impossible and cyanosis and distress result. The baby then cries and by thus beginning to breathe through the mouth the dyspnea is relieved, oxygenation is established and the face and body become flushed. When the child is at ease and the crying ceases, the mouth again closes and the cycle commences again. Richardson refers to this picture as cyclic dyspnea. He also declares that because of inability to breathe through the nose and failure of the mouth to open, some children never breathe at all and are counted among the stillborn infants.

As soon as the infant attempts to suckle, the respiratory distress is greatly accentuated. If the victim survives the first ten days, he learns to breathe through the mouth habitually. This is expedited by the nurse holding the lower lip down a considerable part of the time. A watchful attendant must be at hand constantly until the mouth breathing habit is established. The swallowing of fluid introduced into the mouth by dropper or by spoon is difficult enough. The act of nursing is well nigh impossible.

If the child lives and the condition goes undiagnosed, he inevitably becomes a confirmed mouth-breather. As a rule, he learns to avoid solid food, especially anything requiring to be masticated. There is no way to continue breathing while chewing. There is no way to relieve the vacuum produced in the nasopharynx and ears during the act of swallowing. The embarrassment that he continually experiences can be simulated by anyone pinching his nares shut and attempting to eat a few bites of food. The children who survive are usually undernourished and irritable. They are not well and may be subject to recurring attacks of gastro-intestinal disturbance.

Various symptoms may be noted in these older cases: anosmia, on one or both sides; tears escaping from the nose following weeping; altered vocal resonance; the accumulation and overflow of a peculiarly bluish glairy mucus (Kirby) and erosion of the nares and upper lips; this accumulation may be "poured" out at times by tipping the face downward.

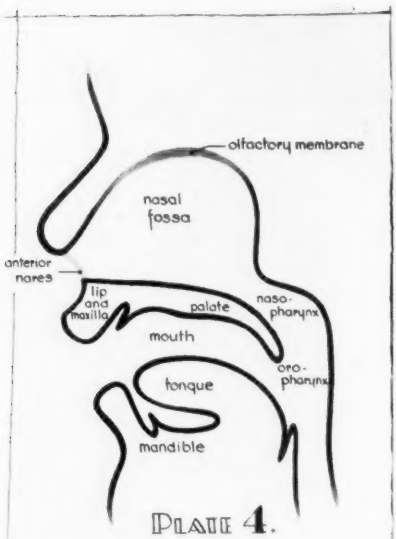


PLATE 4.

The above-mentioned symptoms should lead to a tentative diagnosis of atresia if the possibility of its occurrence is kept in mind. Stinson calls attention to the fact that the dyspnea and cyanosis of atresia resemble the similar symptoms of enlarged thymus and not infrequently lead to a diagnosis of enlarged thymus. Garretson mentions other causes that may produce nasal obstruction and that must be differentiated, including adenoids, syphilitic rhinitis with atresia, congenital synechiae, retropharyngeal tumors, intranasal tumors, intranasal hypertrophies, choanal polypi and foreign body in the nose.

The actual confirmation of the diagnosis may be made as follows: By attempting to pass a probe through the nose, along the floor; by the insertion of the examiner's finger into the nasopharynx to palpate (this being impossible with the average adult finger in the average infantile throat); by the use of a Politzer bag or other rubber bulb syringe in attempting gently but firmly to force air through each nasal passage; by a similar test made by filling the nasal cavities with fluid and observing that it does not flow through (Roth and Geiger); by the use of the nasopharyngoscope. In older children the details of structure and relationships may be studied by the postnasal

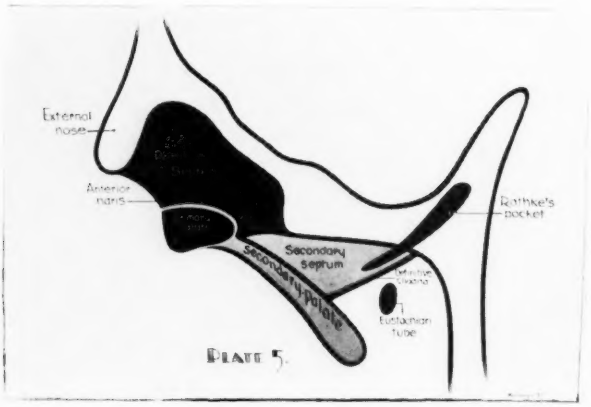
mirror and in roentgenograms, with or without the aid of iodized oil. The most available and valuable means of diagnosis are the use of the rubber bulb syringe and the gentle sounding with a probe.

SURGICAL RELIEF

Surgical relief of the obstruction is the ultimate solution of this problem. Just when and how it is to be done has been the subject of considerable discussion. There are some who believe that when the condition is diagnosed, at or shortly after birth, the child should be taught mouth breathing and later taught to nurse or eat solids. The child may be fed through a small feeding tube, if necessary, at times. The patient is thus to be carried on under general supervision until he attains the age of two years or more. Lebensohn and Roth and Geiger advise deferring the operation until after the child is nine months old, while Garretson urges to "operate at once," if the obstruction is bilateral, as soon as the diagnosis is made. Richardson and Loeb also believe that the operation may be done at an early age, even within the first few days. The question of the time for operation has to do with the bilateral cases, as of course the surgical relief of unilateral obstruction is an operation of convenience and may be deferred indefinitely, according to the wishes of the patient's family and until the best condition of the patient is attained (Jones). The program of delay is to be interrupted only if the child's development or health is not satisfactory. There are others who contend that an immediate or very early operation is best. The decision in a given case might depend on the social circumstances of the family. A family which can supply careful supervision may safely wait for the passage of time. A family without the means to supply proper nursing for the handicapped child would be well advised to have the surgical work done early. It is the author's opinion, in agreement with Phelps, that as a rule the early operation is best under all circumstances. Under ordinary circumstances the child is in as good condition shortly after birth as it is likely to be. Delay is dangerous and the advantage of the increase in size and vigor of the child is of doubtful weight.

If the case is first diagnosed at an age past the suckling period, the moment of the surgical procedure may be chosen in accordance with the physical condition of the child or the convenience of the family. In general, it is best to undertake radical relief without undue delay.

The technic of the surgical operation must vary somewhat with the age of the patient. In the newborn the slightest amount of local anesthetic and vasoconstrictor introduced into the nose is the safest.



(Reprinted from Sir Arthur Keith's "Human Embryology and Morphology." Published by Arnold & Co., London. Page 224, Fig. 215.)

Plate 5. The primitive nasal cavity and primitive choana at the end of the 6th week. The formation of the secondary septum and palate and of the definitive choana is also indicated.

In older children or adolescents a general anesthetic is desirable. Such an anesthetic is attended by hazards and must be given very carefully by a well trained anesthetist. In the hands of an expert, intratracheal anesthesia might be considered the method of choice. At any age a point of great importance is the protection of the posterior nasopharyngeal wall. If the mouth and throat are large enough, nothing is better than the tip of the operator's index finger. This acts not only as a protector but as a guide to the proper removal of the obstruction regardless of what instrument may be used to accomplish it. If the parts are too small for the operator's finger to be used, some other guard must be employed, such as a flexible strip of metal or a bent piece of wood.

The actual removal of the button may be done by a small gouge tapped gently by an assistant, or by some form of trocar, rasp or burr. If the gouge is used, the periphery of the site of removal can be smoothed by a burr or curette. Care must be exercised not to injure the turbinate bones and especially not to injure the structures on the lateral and superior aspects of the choanae. If the operation is done at an early age, no attempt need be made to spare the tiny anterior covering of mucous membrane. In the larger noses this flap may be demarked by an incision extending from the upper outer angle across

the surface of the obstruction to the septum, then passing down the edge of the septum and then laterally along the lower border. The tongue-shaped flap may then be elevated laterally and, after the bony button has been removed, may be carried backward across the edge of the cut surface. This will probably discourage granulations from growing mesialward into the new lumen. After the openings have been established, the posterior portion of the nasal septum should be removed through the mouth and nasopharynx by suitable rongeur forceps. This removal immediately enlarges the choanae by throwing them together and is a safeguard against postoperative closure of the new openings.

This expedient was first utilizied by White in 1914. Late in 1913 he operated in a case of unilateral atresia using the Uffenorde technic. This was by an elevation of the septal mucosa, extending the elevation posteriorly to the obstruction and laterally across it. The bony diaphragm was then removed submucously. Finally the mucosal wall being replaced, the portion overlying the newly made opening was punctured and dilated so as to cover the edges of the choana. This was done to afford immediate membranous protection to the raw surfaces of bone and to discourage granulation. Three months later White found the opening almost closed. He performed a secondary operation and at that time removed a portion of the posterior edge of the septum about the size of a dime. This was successful.

In 1916 White operated in a case of bilateral atresia. In this case also the removal of the posterior portion of the septum was performed. The operation was a success. In reporting these cases in 1918 White states that "it occurred to me that if the raw surfaces were further apart the danger of closure would be greatly lessened"—and therefore he concluded "that the logical thing to do would be to make a perforation in the posterior end of the septum involving the obstructing choana." He was not able at that time to find any case in literature where this method was followed. Later, however, he did discover that Katz, in his textbook, published in 1911-13, speaks, in relation to the operation for the relief of atresia, as follows: "Everything that could be reached was removed from the hinder edge of the vomer. When one makes a sufficiently large opening, and especially has freely removed the posterior edge of the vomer, the after treatment can be carried on with relative simplicity."

Phelps, in 1925, in mentioning the removal of a portion of the posterior edge of the septum as a part of the operation, remarks that in 1910 Dan McKenzie was first to make mention of the removal of the posterior portion of the septum. In reading McKenzie's report

of his case it appears that he performed a submucous resection of the septum as a preliminary operation, acting upon the advice of Dr. Dundas Grant. Some weeks later the choanal diaphragm was cut through. No mention is made of the removal of any of the free border of the septum. In closing the discussion McKenzie further explains that the submucous resection was done because of considerable septal deflection and "to render access to the posterior diaphragm more easy." In an article printed in 1910, Fraser mentions what is probably this same case, as follows: "Dan McKenzie (London) related a case in which the posterior choanal partition was associated with deviated septum. After operation on the septum, the osseomembranous web was broken down with chisel and burr with perfect results." Kirby reports that he operated by performing a submucous resection and removed the malformation through this route. Exactly how this was done is not clear. He also advises the use of a window flap of mucous membrane and the postoperative use of a rubber tube. These cases of McKenzie and of Kirby were cases of unilateral atresia. Stinson, in a unilateral case, where the lateral wall encroached on the operative field and presented a hazard to the direct attack, threw the two nasal chambers together by the removal of a portion of the vomer, thus allowing both sides to utilize one choana, enlarged as it was by the absence of the posterior portion of the septum. In older patients it is quite likely that normal osseous development of the choanal canal has been retarded. For this reason special care must be exercised, in working laterally, to avoid injury to the blood vessels and the nerves in the sphenopalatine canal.

The postoperative care has varied in the hands of different surgeons. Rubber tubes have been introduced, being removed and replaced daily by some, twisted about in situ, but not removed for several days, by others. Or a strip of gauze may be carried into one naris and back through the other. This gauze may be replaced daily by another strip being fastened to it and then gently pulled through by the first strip as it is being removed. A rubber tube or catheter has been used similarly by Mahoney and by Hart and Peeler. In a case of Hubbell, in 1886, block tin was moulded into shape to form tubes to fit the new choanae. These tubes were left undisturbed for several days. In some cases only daily postoperative cleansing was used to insure patulency and proper healing. There is no danger of collapse as the choana is surrounded by a bony framework. The only mechanism of closing is the formation of excessive granulations with gradual narrowing of the surgical openings.

Bourgeois and Poyett remove the offending diaphragm and make no attempt to discourage the granulations, which are very likely to

completely close the surgical opening. After this has taken place they then destroy this secondary membrane by electrocoagulation.

There may be other problems to be solved, such as sinusitis, adenoid vegetations, diseased tonsils or faulty dental arches. The adenoids should be removed at the time of the choanal operation, but the other conditions should be allowed to wait for surgical recovery from the main operation. In most cases it is proper to allow further delay for the improvement that may be possible by treatment after normal nasal ventilation and breathing have been established. Great improvement of pathologic intranasal conditions has been observed after normal ventilation and drainage have been established.

REPORT OF TWO CASES

Case 1.—The case of a little girl with bilateral choanal atresia for whom the author performed a surgical operation in the fall of 1918, was originally reported to the American Laryngological, Rhinological and Otological Society in 1920. It is the present purpose to review the case briefly and to report the present condition of the patient.

The operation was performed in September, 1918, when the child was two years and eight months old and when she weighed fourteen pounds and one-half. A considerable button of the posterior edge of the septum was removed.

It is interesting to note that the attending physician and his consulting surgeon agreed at the birth of the patient that there was absolutely no opening from the nose into the throat. This opinion was confirmed on later occasions by other physicians, but, in spite of this, surgical relief was not urged. The child was allowed to go for more than two and one-half years, having an unhappy and sickly existence. There was dental deformity, interference with breathing and eating, gastro-intestinal disturbance with diarrhea, and malnutrition.

At the age of almost 21 years, the patient was last examined in December, 1936. She has never developed normally, either physically or mentally. Her nasal breathing and condition have been normal, however, except for occasional colds. She has had three attacks of suppurative otitis media. Her tonsils and adenoids were removed when she was about fourteen years of age.

Examination of the posterior choanae with a nasopharyngoscope shows them to be approximately normal in size. There is evidence of a ring, or low ridge, remaining about the periphery of the old obstruction. It is not continuous and is very low except superiorly where, on both sides, there is a sort of valence that hangs down about one millimeter. It is evident that the removal was less complete in this direction. The posterior edge of the septum is rounded and smooth. It is further anterior than normal as is evidenced by looking across it into the opposite side. The line of the ring showing the periphery of the obstruction is seen well back of the edge of the septum. On each side of the throat there are a few adhesions between the choana and the anterior lip of the Eustachian tube, which appear to have arisen from the flap of mucous membrane that was turned backward on the lateral walls.

Case 2.—Another case of bilateral choanal atresia, which is here reported for the first time, was that of a baby girl, six days old, seen on July 18, 1934, in consultation with Dr. R. F. Tatro.

She was suffering from typical cyclic dyspnoea. She could not nurse the breast. She was able to suck for a moment on a rubber nipple, but was fed practically solely with a medicine dropper. The diagnosis was readily established by the use of air pressure from a rubber bulb, and by the introduction of a guarded applicator. The baby did not object to the nares being closed by gentle pressure on the alae. It was felt that surgical interference should be delayed to give opportunity to attempt to build up the infant's strength. Her weight at birth, on July 12, was 7 pounds, 4½ ounces and on the sixth day was 7 pounds, 2 ounces.

Four days later, on July 22, Dr. Tatro reported: "I weighed the baby this morning and it has lost three ounces. It appears stronger and brighter however. I believe that by the latter part of this week or the first of next it would be safe to perform the operation. The baby is getting glucose subcutaneously once or twice a day besides its regular feedings by mouth, so that we are doing all we can to build it up in preparation for the operation." On August 1 the operation was performed.

About thirty minutes before the operation was started, Dr. Arthur George packed both sides of the nose with plain gauze wet with a mixture of equal parts of 2% solution of Butyn and 1 to 1000 solution of Epinephrin. A mattress stay suture, guarded with a piece of rubber tubing, was put through the median raphe at the tip of the tongue and a second stay suture was passed through the border of the soft palate. With a long-handled knife an incision was made on each side through the membrane overlying the bony obstruction in the posterior choanae. These incisions were U-shaped, passing inward along the floor of the nose, upward close to the septum, and outward at the upper border of the obstruction. The flaps of mucous membrane thus outlined were elevated laterally and the mucous membrane of the septum adjacent to the obstruction was retracted forward.

It was found impossible to introduce any finger into the nasopharnyx, even a finger of the surgical nurse. There was no narrow flexible metal strap available, so a protector was improvised. A wooden tongue depressor was split to about half of its original width, the edges were smoothed, and the end bent until it cracked. This turned up about an inch and a quarter of the distal end. It was then introduced behind the uvula and held in place by an assistant. This afforded quite satisfactory defense against instrumental injury of the posterior nasopharyngeal wall.

A small Alexander mastoid gouge was introduced along the floor of the nose. By gentle pressure and turning of the gouge the obstruction was penetrated and the opening reamed to normal size. The debris was removed by bayonet forceps and applicator. A small nasal curette was next introduced and gently rotated so as to trim off any residue around the periphery of the bony diaphragm.

A small rongeur was introduced into the nasopharynx and the posterior edge of the septum seized. A small portion of the septum was excised and removed. Short pieces of rubber catheter, size 12 (French), were introduced into each side of the nose reaching just through the posterior choanae. The posterior end of each tube had been burned and smoothed and an oval fenestrum made in it. These tubes were sutured together externally and anchored to the cheeks with stay sutures held by adhesive tape.

Within twenty minutes the baby had taken a liberal amount of tepid water from a nursing bottle and was sound asleep with her mouth closed. For a few days the rubber tubes were kept in the nose, being slightly turned once or twice a day.

By August 4 the baby was nursing, sleeping and having bowel movements normally. It was gaining about 2 ounces in weight daily. It was taken home on August 8 and the further progress of the patient was satisfactory.

Observations of this patient have been made from time to time. After a few months it was determined that the choanal opening on the right side was apparently normal. The left choanal opening, however, had become somewhat stenosed. Inasmuch as the functions of breathing and eating and the general physical progress were normal, it has been felt best to defer any further surgical attempt to enlarge the left choana. It is planned to do this, however, within the near future.

CONCLUSIONS

1. It is evident that choanal atresia occurs more frequently than the occasional clinical report would indicate.

2. The gravity of bilateral choanal atresia, especially in the newborn, cannot be overemphasized. It is important that the possibility of its occurrence and the method of immediate diagnosis and treatment be given wide publicity in medical literature.

3. Surgical relief should be instituted early when bilateral choanal atresia is diagnosed.

4. The future health and development of the patient depend upon early surgical relief in cases of unilateral atresia, as well as in cases of bilateral atresia.

5. In the surgical technic removal of the posterior portion of the nasal septum is fundamentally important in insuring a free and permanent choanal opening.

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XXXIV

PHARYNGEAL RECONSTRUCTION FOR NASO-PHARYNGEAL STENOSIS: A NEW OPERATIVE PROCEDURE*

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SAN FRANCISCO

Acquired cicatricial stenosis of the nasopharynx, although rarely occurring, is such a perplexing problem to treat that a review of past literature, the surgical progress, some new thoughts and a successful new operative procedure for its correction form the basis of this report.

The difficulties encountered in correcting nasopharyngeal stenosis is evidenced by the great variety of procedures that are advocated, many of which have no practical value. The literature for the past fifty years yields but little material and very few completed case reports until recent years. The probability is, in those reports not completed, that stenosis recurred so that a negative end result was thought not worth recording. The rarity of the condition makes it difficult for many individuals to gather a sufficient number of cases for a thorough study, but much valuable information can be obtained from three excellent articles, one by J. E. H. Nichols, one by J. E. Mackenty, and the more recent review by F. A. Figi of Mayo Clinic.

The majority of nasopharyngeal stenosis cases reported to date are classed under the acquired type, there being only a few that are reported as true congenital malformations. Mackenty subdivides the congenital stenosis into two types: those due to inflammation and those due to malformation. He believes an accurate observation of the lesion under consideration will reveal the class in which it belongs. He records one case which he feels is due to congenital syphilitic stenosis and not congentital pharyngeal malformation. In the congenital malformation cases reported by Hall, Wassermann, Baily, Mackenty and Figi, the ease of division of the attachment of the soft palate to the pharyngeal wall and the tendency of the stenosis

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not to recur markedly contrasts them with the acquired lesions. I have had no experience with the congenital stenotic type, but would surmise that the end results obtained were due to the fine weblike adhesions, the surrounding normal tissue, the small denuded areas and the rapidity of epithelization that takes place under these conditions.

Syphilis is generally considered by most essayists, except possibly Figi, as the most common cause of acquired nasopharyngeal stenosis. Fifty-eight out of sixty-nine cases reviewed by Wright and Smith were said to be due to syphilis. Figi states, "These figures vary so strikingly from the observations in the Mayo Clinic that I cannot but wonder whether a large proportion of the cases were not seen in charity clinics, where the patients, because of their own indifference, failed to secure treatment during the stage of active pharyngeal ulceration." He reports five out of eighteen cases as being due to syphilis. Wright and Smith in their review then mention as causative agents, in the order of their frequency, trauma, diphtheria, tuberculosis, congenital anomalies and inflammatory lesions. These correspond with the more recent experience of Mackenty and Figi. Mackenty suggests a "cicatrizing diathesis" as the determining factor to explain some stenosis after inflammation or trauma. He states, "I believe it would be next to impossible to produce experimental atresia by trauma." It is a well known fact that certain individuals while effecting cutaneous healing develop hypertrophied scars or large keloids and are spoken of as having a keloid tendency. The reaction that occurs in some mucous membrane surfaces is probably of the same character and does not occur more frequently only because of the inherent anatomy of the mucosa.

The symptoms presented in the case I am reporting were those of complete nasal obstruction, change of voice, anosmia, impaired hearing and respiratory difficulty on exertion. The symptoms complained of for a given case depend largely upon the degree of pharyngeal obstruction present. Chronic sinusitis, deafness, developmental changes in the fauces and contour of the nasopharynx, as well as pulmonary complications have been reported. Figi reports that 50 per cent of his cases were under twenty years of age and states, "Since trauma and hereditary syphilis are the great offenders, this is to be expected."

The diagnosis is usually apparent by inspection, but a complete investigation of the upper respiratory passage should be done to rule out choanal atresia or nasopharyngeal neoplasm. This examination will also acquaint one with the degree and type of stenosis at hand. Most of the reported cases had a small midline opening in the vicinity of the uvula and to this my case was no exception.

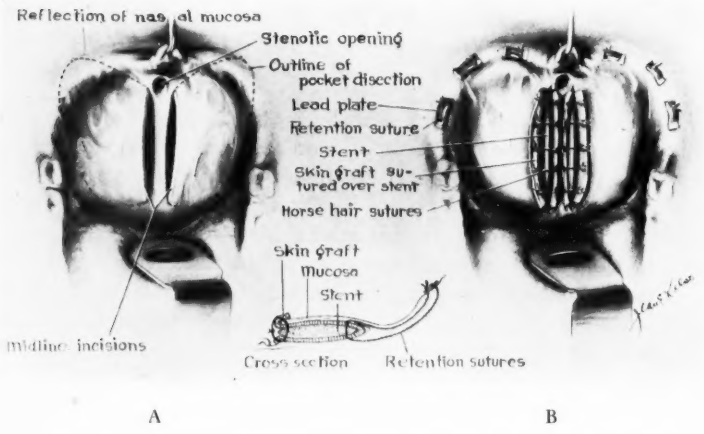
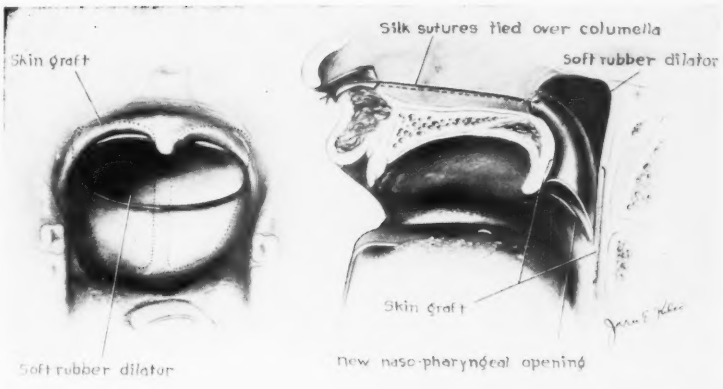


Fig. 1. *A.* Stenosis of the nasopharynx showing incisions and dissection. *B.* Stent impressions covered with skin grafts sutured into position. The peripheral retention sutures are most important.

The prognosis depends upon the degree and type of stenosis, the amount of scar, the causative agent, but principally the method of treatment elected. The earlier writings on the prognosis and treatment of stenosis of the nasopharynx were extremely pessimistic but, with the advent of Nichols' paper on the seton method and Mackenty's plastic procedures, the reports are more optimistic. The prognosis seems better when using the seton method, as the Mackenty plastic procedure depends on normal mucous membrane, which is rarely present, so he too is inclined to the seton method in certain cases. Figi reports favorably on the Nichols seton method, saying: "Numerous procedures were tried with only fair success, but in the last six cases the seton method of Nichols were employed and these patients obtained complete relief of symptoms."

There is a stenosis of a hollow muscular tube due to the loss of epithelium and the replacement with dense contracting scar tissue. The tube is fixed in the posterior half, open and movable (the soft palate) in its anterior half. The stenotic ring of scar is thicker at its base than about the center point. The stenosis occurs due to a loss of epithelium in the nasopharyngeal wall and the posterior velum palati. The scar tissue that forms in the process of healing automatically causes adhesions and closes the nasopharyngeal opening. The soft



C D

Fig. 2. *C* and *D*. Anterior-posterior and lateral views of the new nasopharyngeal opening with dilators in place.

palate and pharyngeal muscles in closing the nasopharynx normally act as a sphincter unit so that this circular pull plus the longitudinal and constrictor fibers of the constrictor muscles of the pharynx pull the opening downward, backwards and together. The greater pull is in this direction due to the pharyngeal wall being stationary and the soft palate and lateral pharyngeal walls more movable. Dr. M. F. McCarthy of Cincinnati has recognized this factor and has used a pharyngeal muscle excision and transplantation to offset the muscle pull. The anatomy of the part and the muscle pull, as well as the cicatricial contraction are contributing factors as to why stenosis occurs at this point, but the stenosis proper is caused by the approximation of the denuded surfaces. Nichols was the first to apprehend the true pathologic nature of the condition and to attempt to correct it from a scientific point of view. He states, "All operations and methods have one defect in common, fatal to their success and which allow the reproduction of the original adhesions. The defect is, that no matter how deep the incision has been made or how much tissue has been excised, the cicatricial tissue steadily advances in the process of healing from the bottom (angles) of the cut." I would add that this process of scar tissue reformation can only be stopped by a limiting membrane of epithelium.

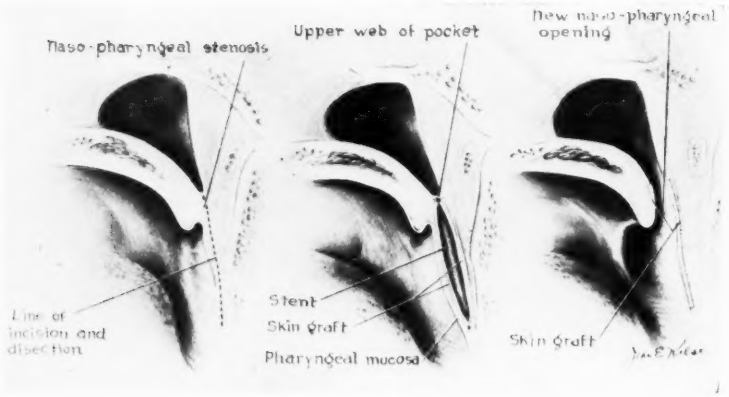


Fig. 3. *E.* Lateral view of nasopharynx. 1. Nasopharyngeal stenosis. 2. Pocket dissected with Stent and skin grafts in position. 3. Open nasopharynx with skin grafted area.

The numerous procedures advanced for relief of nasopharyngeal stenosis are an indication of the inadequacy to effect a cure or even a moderate degree of relief of symptoms. This is due not only to the character of the pathology and the inaccessibility of the surgical field, as suggested by Mackenty, but also to the failure to apply the basic principles of reconstruction surgery in the majority of procedures. The pathologic problem may well be understood, but unless a method of attack is devised that will adequately correct it the efforts can only result in failure.

Any surgical procedure or mechanical contrivance devised to correct stenosis of the nasopharynx must ultimately fail in effecting a cure unless such procedure supplies epithelium immediately to the denuded surfaces as does Mackenty, Roes or Axhansen procedures, or delayed as in the seton method of Nichols, wherein Nature supplies some covering after a sufficient waiting period.

1. Dilatation without operation.
2. Dilatation with incision, as advocated by Ellsberg, Diffenbock, J. Guisez, Conetoux and Kuhn, Isaacs and others.
3. Incision with acid cauterization, as done by Teets.
4. Diathermy, as practiced by Bourgeois and Royet.

These methods are mentioned only to be condemned, for in failing to comply with the essential requirement for success, supplying epithelium to the denuded surfaces, they defeat themselves.

COMPARISON OF WORKABLE PROCEDURES

1. Axhansen's operation of tracheotomy, pharyngotomy and external plastic flaps is too radical, deforming and not necessary to correct nasopharyngeal stenosis.

2. Mackenty's plastic reconstruction is deficient in that it supplies a covering to only one surface; epithelization is not complete at the base of the lateral angles, a prerequisite to success, according to Nichols. At best it is only useful in minor degrees of stenosis, for Mackenty states, "When the small opening in the center of the palate is absent this signifies a more extensive atresia and makes the operation more difficult and the outlook less hopeful since a larger flap is needed to cover the defect. This is too often not available." He further adds, "If nothing but scar tissue be on the posterior pharyngeal wall, if the atresia above is extensive, if the whole pharynx is contracted to the center, and especially if syphilis (the greatest single factor reported) is the cause we have almost a hopeless condition on our hands." In speaking of Nichols' seton procedure, he states, "I think, so far it is the best operation in many cases." Mackenty's second procedure of creating a cleft palate is too radical, unnecessary and not aimed at correcting the original defect but places an additional burden of a cleft palate on an already deformed upper respiratory passage.

3. Practically all of the deficiencies in the Mackenty operations again present themselves in the Roe plastic operation. Mackenty, speaking of Roe's method, states, "It has never been proved off paper."

4. Nichols' seton method. This has been the most successful method to date, as shown by the reports of Chase, Figi, Leland, Nichols and others, but it lacks many of the features necessary for pharyngeal reconstruction. It does not supply epithelium or reconstruct the existing defect but effects its results by several scar tissue channels that do not permit the adhesions to reform at these points; the thicker the base of the stenosis, the longer it takes for the channels to be formed and eventually the more the denuded surface. All of the denuded areas between the seton scar channels must of themselves either readhere or form scar and contract, as do the seton channels, for the physiologic reaction of cicatricial tissue is to contract. The

F

Fig. 4. *F.* Preoperative condition with practically a complete stenosis of the nasopharynx.

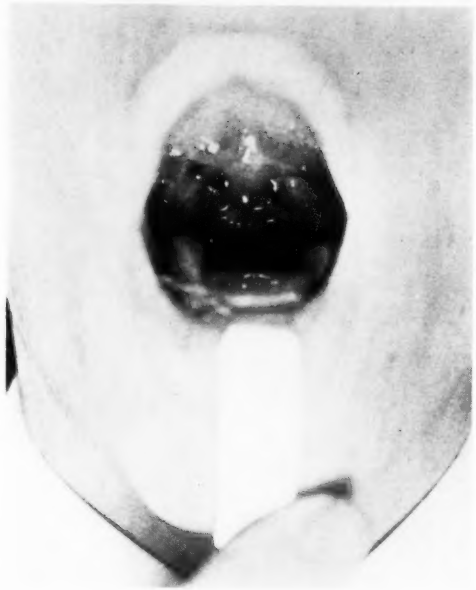


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Fig. 5. *G.* Postoperative result. Normal soft palate-palatal arch present. White patches on pharyngeal walls are the skin grafts.

seton method does not correct any contraction of the pharyngeal wall or the soft palate that has occurred, but merely re-establishes a small slit-like passage between the soft palate and pharynx, thus giving a small functional opening.

The thought of using skin grafts to correct nasopharyngeal stenosis is not new, but the method of application and the successful termination of the case apparently is. Mackenty in 1926 states, "The use of skin and mucous membrane transplants placed against the juxtaposed raw surface of the released palate has been advocated. No cases have been brought forward attesting to the virtues of this method, hence I am of the opinion that these reports should be stigmatized as surgical vaporings, of which there is no dearth in the literature." Possibly the vaporings have finally condensed. Figi, in 1929, reports having observed eighteen cases at the Mayo Clinic, fourteen of which were operated upon by various methods and two of these having dilatation and skin graft, with the statement "that they did not receive permanent benefit." These two are the only cases I have found reported in the literature on the application of skin grafts for the correction of nasopharyngeal stenosis.

From my own case and the description and pictures of the other reported cases, I feel that the *epithelial pocket method* is applicable to all cases of acquired nasopharyngeal stenosis. The problem is essentially the same, except one must vary his technic according to the amount of scar and stenosis present.

REPORT OF A CASE

CASE 1.—My patient was a young Italian boy, referred by Dr. C. Bricca, 18 years of age; negative blood Wassermann; no history or evidence of cutaneous keloids; several attacks of tonsillitis previous to July, 1934.

The chief complaints were: (1) Complete nasal obstruction; (2) loss of sense of smell; (3) change in voice quality; (4) mucus in throat; (5) decreased hearing.

In July, 1934, tonsils and adenoids were removed, and in August, 1936, he was told by the same physician his adenoids had regrown, so on August 15, 1936, an adenoidectomy was performed. Patient states that he had a sore throat for ten days, then four days later he developed considerable mucus in his throat and marked difficulty in nasal breathing. Three weeks later (September, 1936) he was operated upon under general anesthesia to open his throat. He was then treated in the office for two weeks with daily applications of silver nitrate solution. At the end of two weeks his throat grew together and he could not breathe through his nose. He had had his throat incised four times under local anesthesia to no avail. He has had no additional treatment but has not been able to breathe through his nose since October of 1936.

Throat examination (January 7, 1937): The anterior and posterior palatal arches were obliterated. The tonsils were removed. There was a small 3 mm. opening into the nasopharynx directly behind and at the base of the uvula. The lateral aspects of the pharynx and the posterior border of the soft palate were heavily scarred and contracted in a cone shape. This whole arch was contracted downwards and backwards and firmly attached by dense cicatricial tissue to the posterior pharyngeal wall. The throat was filled with a thick, tenacious mucus and the patient was unable to breathe through the small opening in the center because the uvula acted as a shut-off valve.

Operation: The patient was operated upon under general anesthesia in the Rose position (as in cleft palate cases). A Davis mouth gag gave splendid exposure of the operative field. The uvula was retracted anteriorly and a 1½-inch incision was made vertically in the pharyngeal wall, starting just lateral to the nasopharyngeal opening, at its middle, and continuing inferiorly for 1½ inches. Immediate lateral retraction of the pharyngeal covering was noted. With a right angled scissors blunt dissection was carried laterally as far as possible. In the superior part the dissection was carried just inside the nasal lining of the stenosis. The dissection was much wider in its superior than in its inferior part. An impression of the pocket was made in Stent (dental modeling compound). The impression was 1½ inches long, ½ inch thick and as wide as the pocket permitted, being widest superiorly. Three holes evenly spaced were then made through the Stent. A thin one-piece Thiersch graft was taken from the inner arm and wrapped, raw surface out, around the Stent. Three medium silk sutures were placed through the holes in the Stent perforating the skin graft on both sides. These then were directed through the pocket on needles and brought out through the pharyngeal wall and soft palate at the lateral, superior and medial parts of the pocket, and tied over fine rubber tubing. Horsehair sutures were then placed through the overlapping skin graft, the anterior flap of the pocket, over the Stent, through the posterior pocket flap, through the overlapping skin graft on the other edge and loosely tied; thus completely covering the denuded area with skin. The same procedure was carried out on the opposite side. For twenty-four hours nothing was given by mouth, resorting to intravenous glucose and subcutaneous saline. Liquids and gruels were then permitted. One week later the Stent was first removed and then under geenral anesthesia and by sharp dissection the fine web of the superior pole of the pocket was cut from the old central opening behind the uvula as far laterally as necessary. A curved cone shaped rubber dilator, 40 mm. in diameter, shorter on its anterior than posterior surface, to limit irritation from swallowing, was introduced with the wide end of the cone towards the mouth. This was held in place by two silk sutures brought through the nostrils and tied over the columella. The sutures were threaded over fine rubber tubing to protect the nasal mucosa and the columella skin. Glucose and saline were given for twelve hours and then a high caloric liquid diet. Gruels and soft foods were permitted after four days. He remained in the hospital one week after the second operation and then reported to the office. The dilator was left in place sixteen days and then removed, as the patient developed influenza. Finger dilatation by the patient and gradual dilatation at the office was then done daily. Finger dilatation, talking exercises and soda bicarbonate gargles have been persisted in to date, but office dilatation is now done only occasionally.

At the present time, four months after operation for the opening of his throat, the patient has a complete cure of his symptoms, a fairly normal throat with the exception of patches of skin in the posterior and lateral pharyngeal areas and an opening 30 mm. in diameter into his nasopharynx.

COMMENT

1. This method reconstructs the whole stenotic area with epithelium, whereas the seton method just establishes an opening through the existing scar.

2. This type of a skin graft properly immobilized will take 100 per cent of the time, even in the mouth.

3. I advise against the use of free mucosa, as it is difficult to obtain and uncertain in its take.

4. It is necessary to overcorrect the defect while making the epithelial pocket because contraction will occur.

5. Continual soft rubber dilatation after opening is imperative for at least two weeks, as the base of the skin grafts shrink and daily dilatation thereafter by the patient and physician should be persisted in. Continual daily elastic-like stretching will give far better results than occasional dilatation.

6. To the men whose reports have preceded my paper, particularly Nichols, Mackenty and Figi, I am extremely grateful for the vast store of knowledge revealed.

7. I realize I have been dogmatic in some of my statements and that one case does not prove a theory. However, I am so firmly convinced that the epithelial pocket method answers the physiologic problems present that I offer it as an aid in these difficult cases and only hope it will be thoroughly tried.

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XXXV

THE CAUSE OF DIZZINESS IN HEAD INJURIES:

A VESTIBULAR TEST STUDY IN SIXTY-SIX PATIENTS*

MARK ALBERT GLASER, M.D.

LOS ANGELES, CAL.

The cause of dizziness in head injuries is a question which has aroused considerable controversy. The value of vestibular tests as a diagnostic adjunct in cases of head injury, as well as their ability to prognosticate the duration of dizziness, has been the subject of extended discussion. Mygind,[1] Gottlieb,[2] Fletcher,[3] Hofmann,[4] Brunner,[5] Linthicum and Rand,[6] Stier,[7] Zachs,[8] and Grove.[9] The sixty-six cases here presented have been analyzed in an attempt to answer these questions, as well as to add supplementary data of importance.

The vestibular tests were first introduced by Robert Bárány,[10] who further demonstrated their value for the diagnosis of intracranial lesions. For this outstanding work he was awarded the Nobel Prize in 1915. The popularization and widespread clinical application of the tests we owe to Jones and Fisher.[11] Among the contributions of these men were evidence that distinct pathways extended intracranially from each individual semicircular canal; the fact that pathways of nystagmus and vertigo were separate and that perverted nystagmus was pathognomonic of an intracranial lesion; the description of a phenomena complex associated with angle tumors and other lesions; and the creation of a definite technic, together with a chart for the proper recording of these findings. E. R. Lewis[12] demonstrated the co-working of the semicircular canals, devised numerous models for their study, and extensively described the mechanics of the end organ.

In order to qualify for aviation, the applicants were required to pass a vestibular examination. Lyster and Jones[13] established a routine technic for this examination, which was immediately adopted by the United States Air Service. Since then numerous articles have appeared upon the importance of neuro-otology in aviation medicine, the latest and most important contribution being that of Jones and Ocker[14] upon blind flying.

*Read before the Eye, Ear, Nose and Throat Section of the California Medical Association at the sixty-fourth annual session, Yosemite National Park, California, May 14, 1935.

In 1923 Wishart[15] analyzed eleven cases of brain tumor and further demonstrated the value of these tests. In 1925 Jones and Spiller[16] discussed the central vestibular pathways, while in the same year Jones and Ingham[17] very clearly resumed the problems arising in neuro-otology. In 1926 Grant and Fisher[18] reviewed 116 cases of verified brain lesions; from these they drew up a group of vestibular complexes for cerebellar lesions, supratentorial lesions, and they verified the cerebellopontile angle syndrome. In 1929 Fisher and Glaser[19] reviewed 139 cases of brain lesions and pointed out that vestibular tests were valueless in the determination of increased intracranial pressure, differentiated the supra- from the sub-tentorial lesions, demonstrated signs of laterality of supra- and sub-tentorial lesions; described a syndrome for mid-central supratentorial lesions; described group findings associated with mid-line cerebellar lesions; verified the cerebellopontile angle syndrome of Jones and Fisher, and described complexes most frequently associated with cerebral lobe lesions. Subsequently, Zimmermann and Tchernychew,[20] Eagleton,[21] Keeler,[22] Winston,[23] Shuster,[24] and Tweedie[25] pointed out the value of these tests as a diagnostic adjunct to the neurologic examination. In addition, neuro-otologic studies in epilepsy were carried out by Jones[26] and Langdon;[27] investigations of the vestibular apparatus in neurosis and psychosis were made by Schilder;[28] while Barré[29] did work on vestibular disorders in intracranial hypertension.

Trauma to the head may injure the peripheral vestibular apparatus or damage the central pathways. Concussion of the labyrinth may result not only from direct blows upon the head, but also from injuries in other parts of the body, the latter due to transmission of the percussion wave along the spinal canal. Various types of end organ pathology may exist and, under certain circumstances, the labyrinthine capsule may explode; in other cases, the nerve constituents enclosed within the labyrinthine capsule may be damaged or destroyed. Degeneration of the nerve or the organ of Corti may occur not only through the impact of fluid, but also through hemorrhage, Risk,[30] Halsz,[31] Rhese,[32] Passow,[33] Stenger[34] and Amberg.[35] Animal experiments undertaken by Voss[36] showed consistent hemorrhage, especially in the perilymphatic spaces of the cochlea and between the branches of the cochlear nerve, whereas the vestibular semicircular canal apparatus remained untouched.

In the present series of 325 instances of head injury, followed from one to eight years after trauma, 80 per cent of the entire series complained of subjective symptoms and 21 per cent revealed objective neurologic signs. Headaches occurred in 67 per cent of the cases, dizziness of some kind in 60 per cent. Vestibular tests were performed

in sixty-six of these patients, the majority by Isaac H. Jones; others by Fred Linthicum and J. J. Pressman. These tests were never carried out in the acute phase of the injury, but were performed during varying lengths of time following trauma. In 76 per cent of the patients abnormalities were demonstrated by the vestibular test, which compares favorably to the 21 per cent of objective signs found upon neurologic examination. These patients have been subjected to a careful analysis in order to determine the value of the vestibular tests in the diagnosis and prognosis of head injuries, as well as whether any relationship existed between the complaint of dizziness and the variations noted upon vestibular study.

In this series of patients 40 per cent revealed central lesions, 36 per cent had end organ lesions and 24 per cent demonstrated normal vestibular responses (Chart 1). Fifty-six per cent of this group had different types of skull fractures, while 87 per cent had varying periods of unconsciousness lasting momentarily to one month. Forty-four per cent did not have a skull fracture and 13 per cent were never unconscious (Charts 2 and 4). Dizziness has been arbitrarily divided into four types: true vertigo, postural dizziness, general dizziness and a combination of postural and general dizziness. Dizziness was entirely absent in 25 per cent of this series.

Dizziness has been a subject of extensive discussion and the types associated with head injuries have been described by Grove,[37] Brain,[38] McMurray[39] and Hubby.[40] Vertigo may be defined as a subjective sensation in which objects, though stationary, appear to move in various directions, or the patient himself may have the sensation of turning about objects. It thus involves a subjective sensation of disturbed relations with enviroment in some definite plane, vertical, horizontal or oblique (Lewis[41]). This is caused by the slightest irritation of the peripheral portion of the vestibular nerve, which includes the end organ and the extra-cerebral portion of the eighth nerve. The sensation is associated with extreme disability and is accompanied by nystagmus, falling, a staggering gait, nausea and vomiting. Vertigo may be caused by tumors, infections, toxins, etc. Vertigo from end-organ disturbance is short lived, approximately lasting six weeks, and then disappears. With this particular point in view, careful histories of these patients were taken and in only one could a history of true vertigo be elicited. The majority of these cases were under personal neurosurgical observation from the onset of the injury, though the neuro-otological tests were always performed after the acute phase had subsided.

The postural type of dizziness is of a milder degree and lasts from a few seconds to several minutes. It is usually caused by a

1. VESTIBULAR DIAGNOSIS

	CASES	PERCENT
I. CENTRAL LESIONS 10 of these have cochlear involvement.	26	40
II. ENT ORGAN LESIONS	24	36
III. NORMAL VESTIBULAR	16	24

2. TYPES OF SKULL FRACTURE

	CASES	PERCENT
I. NONE	28	44
II. BASE	12	66
III. VAULT	9	13
IV. DEPRESSED	8	12
V. VAULT & BASE	6	9

3. TYPES OF DIZZINESS

	CASES	PERCENT
I. POSTURAL DIZZINESS	31	47
II. NO DIZZINESS	17	25
III. GENERAL DIZZINESS	15	22
IV. COMBINED DIZZINESS	3	4
V. VERTIGO	0	0

4. UNCONSCIOUS PERIOD

	CASES	PERCENT
I. NOT UNCONSCIOUS	9	13
II. MOMENTARY	10	15
III. ONE-HALF HOUR	12	18
IV. 1/2 to 2 HOURS	5	7
V. 2 to 12 HOURS	14	13
VI. 12 to 48 HOURS	5	7
VII. 48 HOURS to 1 MONTH	8	12

change of posture, such as stooping, shaking the head from side to side, or up and down, and arising from bed. In addition to this postural dizziness, the same patient may have the same type of dizziness brought on by sudden changes in temperature, exposure to sunlight, excitement, riding in elevators, exertion, the use of alcohol or tobacco, and gastro-intestinal disturbances. The dizziness is not an actual rotation, but is a sense of unsteadiness, instability, or giddiness accompanied by a feeling of wavering or swaying, objects before the patient's eyes become black or gray. These patients fear heights, believing they will fall, but rarely do, yet they frequently do stagger. Such a type of dizziness proves throughout repeated examination and frequently exists over a period of years. Thirty-one patients, or 49 per cent, complained of this type of dizziness (Chart 3).

Generalized dizziness is never "clear-cut," according to the patient's description, for they describe a type which is a combination of true vertigo and postural dizziness, but yet with many variations present. The general form is of longer duration, lasting for many hours or even days. The patient's complaint is entirely disproportionate to the type of dizziness which actually could exist with the routine of life he describes himself as living. Upon repeated observations, over a period of months to years, the complaint continuously varies and rarely does the patient give the same description. In this series of cases generalized dizziness occurred in fifteen patients, or 22 per cent of the entire group (Chart 3).

5. VESTIBULAR REACTIONS PRESENT IN ENTIRE SERIES 66 CASES

	CASES	PERCENT
I. SPONTANEOUS VERTICAL NYSTAGMUS		
Present	3	4
Absent	63	96
II. SPONTANEOUS PAST-POINTING		
Normal	63	96
Exaggerated	3	4
III. TURNING		
Nystagmus		
None	2	3
Normal	23	35
Exaggerated	7	10
Subnormal	34	52
Perverted	0	0
Not given	0	0
Motion Sensing		
Normal	26	40
Exaggerated	9	14
Subnormal	23	35
None	1	1
Not given	7	10
IV. CALORIC		
Vertical Canals		
Normal	25	38
Exaggerated	1	1
Subnormal	34	52
None	4	7
Perverted	1	1
Not given	1	1
Horizontal Canals		
Normal	20	31
Exaggerated	0	0
Subnormal	40	61
None	1	1
Perverted	4	6
Not given	1	1
Motion Sensing		
Normal	17	26
Exaggerated	0	0
Subnormal	33	51
None	7	10
Not given	9	13
Constitutional Responses		
Normal	12	18
Absent	27	41
Exaggerated	2	3
Subnormal	22	34
Not given	3	4
Past-pointing		
Normal	33	51
Exaggerated	8	12
Subnormal	22	34
Not given	2	3

A combined form of true postural dizziness, associated with the general type, is occasionally present and leads to further confusion in actual complaints. In the present group three patients complained of combined dizziness, or 4 per cent of the entire series (Chart 3).

The vestibular charts of the sixty-six patients were analyzed minutely to determine whether any particular vestibular complex would be diagnostic of the postconcussion type (Chart 5). A survey of Chart 5 indicates spontaneous nystagmus was absent and also that spontaneous past pointing was normal in 96 per cent of the patients. Nystagmus after turning was normal in 35 per cent and subnormal in 52 per cent. Motion sensing, after turning, was normal in 40 per cent, subnormal in 35 per cent, exaggerated in 14 per cent. After douching, the vertical canals were normal in 38 per cent and subnormal in 52 per cent. The horizontal canals were normal in 31 per cent and subnormal in 61 per cent. Motion sensing, after douching, was normal in 26 per cent and subnormal in 51 per cent. The constitutional responses were normal in 18 per cent, absent in 14 per cent and subnormal in 34 per cent. Past pointing, after douching, was

6. ANALYSIS OF CASES WITH NORMAL VESTIBULAR RESPONSES 16 CASES

	CASES	PERCENT
I. SKULL FRACTURE		
1. Absent	9	56
2. Depressed	3	18
3. Vault	0	0
4. Base	2	12
5. Vault and Base	2	12
II. DIZZINESS		
1. Vertigo	0	0
2. Postural	5	31
3. General	6	38
4. Combined	1	6
5. Absent	4	25
III. SEVERITY OF INJURY		
1. Not unconscious	6	38
2. Momentarily	3	18
3. Unconscious period		
a. 1/2 hour	1	6
b. 1/2 to 2 hours	0	0
c. 2 to 12 hours	4	26
d. 12 to 48 hours	2	12
e. 48 hours to 1 month	0	0

7. Measurements with Audio-Amplifier

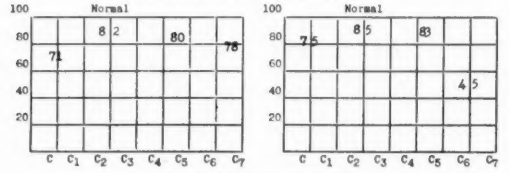
RIGHT EAR. LEFT EAR.

I. Amplifier.

Voice, confused ____, distinct 97%. Voice, confused ____, distinct 100%

Whisper, percentage hearing ____. Whisper, percentage hearing ____

II. Percentage Hearing for Tones.



III. Upper Limits of Audibility.

16,384 d.v. 14,000 d.v.

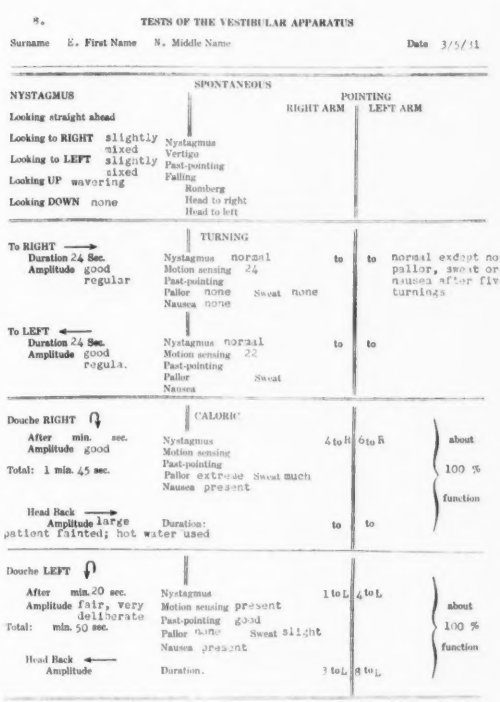
IV. Bone Conduction.

128 94 . 256 103 . 512 112 128 97 . 256 106 . 512 112

normal in 51 per cent, exaggerated in 12 per cent and sub-normal in 34 per cent. Hence, the figures indicate that a predominating picture of a postconcussion syndrome is one of normal spontaneous responses; after turning, normal and subnormal nystagmus; after douching, normal and exaggerated responses from the vertical canals; normal and subnormal responses from the horizontal canals; normal and subnormal motion sensing, absent and subnormal constitutional responses, normal and subnormal past pointing.

NORMAL VESTIBULAR RESPONSES

Sixteen (24 per cent) of the group had normal vestibular responses (Chart 6); skull fracture was absent in 56 per cent; the



S. TESTS OF THE VESTIBULAR APPARATUS

Surname E. First Name N. Middle Name Date 3/5/31

SPONTANEOUS

NYSTAGMUS

Looking straight ahead

Looking to RIGHT slightly mixed

Looking to LEFT slightly mixed

Looking UP wavering

Looking DOWN none

Nystagmus
Vertigo
Past-pointing
Falling
Romberg
Head to right
Head to left

POINTING

RIGHT ARM | LEFT ARM

TURNING

To RIGHT →
Duration 24 Sec.
Amplitude good
regular

Nystagmus normal to
Motion sensing 24
Past-pointing
Pallor none Sweat none
Nausea none

to normal except no pallor, sweat or nausea after five turnings

To LEFT ←
Duration 24 Sec.
Amplitude good
regula.

Nystagmus normal to
Motion sensing 22
Past-pointing
Pallor Sweat
Nausea

to

CALORIC

Douche RIGHT
After min. sec.
Amplitude good
Total: 1 min. 45 sec.

Nystagmus 4 to R
Motion sensing
Past-pointing
Pallor extreme Sweat much
Nausea present

6 to R

about 100 % function

Head Back →
Amplitude large
patient fainted; hot water used

Duration:

to

to

Douche LEFT
After min. 20 sec.
Amplitude fair, very deliberate
Total: min. 50 sec.

Nystagmus 1 to L
Motion sensing present
Past-pointing good
Pallor none Sweat slight
Nausea present

4 to L

about 100 % function

Head Back ←
Amplitude

Duration. 3 to L

8 to L

remaining 44 per cent had varying types of fractures. Postural dizziness was present in 31 per cent, generalized dizziness in 38 per cent, while 25 per cent of these patients, though suffering different degrees of head injuries, did not complain of dizziness Thirty-eight per cent of these patients were never unconscious, 18 per cent were momentarily so, and 26 per cent so from two to twelve hours, while the remainder had varying periods of unconsciousness. The salient features of this group were the absence of skull fracture and the great percentage of patients who did not lose consciousness. Nevertheless there were patients who had severe injuries producing hemiplegia, aphasia, insanity, and continuous dizziness which resulted in a lengthy disability. Yet in those same patients whose clinical examination indicated severe brain damage, normal vestibular responses were also present. From these facts, therefore, it is evident that a serious head injury may exist without the involvement or irritation of the peripheral or central vestibular pathways.

9. ANALYSIS OF CASES WITH END-ORGAN LESIONS 24 CASES

	CASES	PERCENT
I. SPONTANEOUS VERTICAL NYSTAGMUS		
Present	0	0
Absent	24	100
Not given	0	0
II. SPONTANEOUS PAST-POINTING	0	0
III. TURNING		
Nystagmus		
None	1	4
Normal	7	29
Exaggerated	1	4
Subnormal	15	63
Perverted	0	0
Not given	0	0
Motion Sensing		
Normal	5	20
Exaggerated	1	4
Subnormal	13	56
None	0	0
Not given	5	20
IV. CALORIC		
Vertical Canals		
Normal	8	33
Exaggerated	0	0
Subnormal	13	56
None	3	12
Perverted	0	0
Not given	0	0
Horizontal Canals		
Normal	7	29
Exaggerated	0	0
Subnormal	16	67
None	1	4
Perverted	0	0
Not given	0	0
Motion Sensing		
Normal	6	25
Exaggerated	0	0
Subnormal	13	55
None	3	12
Not given	2	8
Constitutional Responses		
Normal	5	20
Absent	8	34
Exaggerated	0	0
Subnormal	11	46
Not given	0	0
Past-pointing		
Normal	9	38
Exaggerated	4	16
Subnormal	11	46
Not given	0	0

REPORT OF A CASE

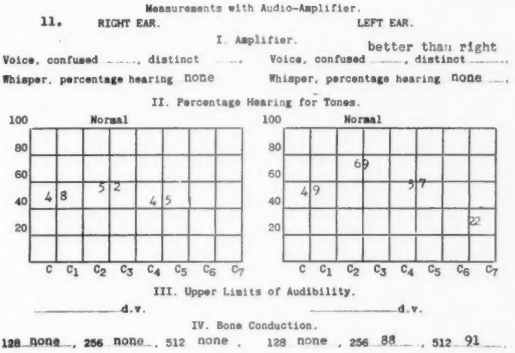
CASE 1 (Normal Vestibular Tests).—E. N., aged 21 years, April 6, 1929, was struck on the head by an iron beam. He received a compound comminuted fracture of the left parietal region and remained unconscious for two days. The fracture was immediately elevated, but with considerable loss of brain tissue. Upon regaining consciousness, the patient had a complete motor aphasia with a paralysis and paresthesia of his right arm and leg. He complained of nervousness, weakness, choking sensations, as well as gastro-intestinal upsets. The paralysis and aphasia gradualy improved. Under observation over a period of five years, residual symptoms still persisted. The young man, upon examination, was ambulatory; blood pressure was 140 75; temperature, 98.6; weight, 156 pounds; height, 6 ft. 1 in.

Physical examination was essentially negative, except for a chronic tonsillitis.

Neurologic examination revealed a cranial defect about the size of a silver dollar situated in the left parietal region. The cranial nerves were entirely negative. Patient had a moderate motor aphasia; the grip in his right hand was 70, left hand 160. The reflexes on the entire right side of his body were much more active than those on his left. There was a definite spasticity of his right arm, hand and fingers, with clumsiness and limited motion. X-rays of the skull showed a cranial defect in the left parietal region.

10. SYMPTOMATOLOGY IN END-ORGAN LESIONS 24 CASES

	CASES	PERCENT
I. SKULL FRACTURE		
1. Absent	16	67
2. Depressed	2	8
3. Vault	1	4
4. Base	1	4
5. Vault and Base	4	17
II. DIZZINESS		
1. Vertigo	0	0
2. Postural	12	51
3. General	6	25
4. Combined	2	8
5. Absent	4	16
III. SEVERITY OF INJURY		
1. Not unconscious	4	16
2. Momentarily	3	13
3. Unconscious period		
a. 1/2 hour	7	31
b. 1/2 to 2 hours	4	16
c. 2 to 12 hours	4	16
d. 12 to 48 hours	1	4
e. 48 hours to 1 month	1	4



Auditory tests (Dr. Isaac Jones) (Chart 7) revealed a perfectly normal cochlea with slight involvement of each middle ear, presumably due to a chronic tonsillitis. Vestibular test (Chart 8) showed a normal function of the vestibular portion of each ear. There was no involvement of the vestibular intracranial pathways. After douching the right ear the patient fainted. This was suggestive of a general hyperactivity of the central nervous system. Even though this accident was one of great severity, the internal ear and the intracranial vestibular pathways escaped damage.

END ORGAN LESIONS

Twenty-four patients (36 per cent) had end organ lesions. In this entire series spontaneous vertical nystagmus was absent and spon-

12. TESTS OF THE VESTIBULAR APPARATUS

Surname J. First Name S. Middle Name Date 6/9/30

	SPONTANEOUS	POINTING	
NYSTAGMUS		RIGHT ARM	LEFT ARM
Looking straight ahead none		3 to R	4 to R
Looking to RIGHT none	Nystagmus		
Looking to LEFT none	Vertigo		
Looking UP none	Past-pointing		
Looking DOWN none	Falling		
	Romberg		
	Head to right		
	Head to left		
	TURNING		
To RIGHT →			
Duration 15 Sec.	Nystagmus	to	to
Amplitude good	Motion sensing 7 seconds		
Good; 14 seconds	Past-pointing		
	Pallor Sweat		
	Nausea		
To LEFT ←			
Duration 9 Sec.	Nystagmus	to	to
Amplitude much smaller	Motion sensing 9 seconds, very indefinite; he gets		
Similar; 13 seconds	Past-pointing very little of actual response, and		
	Pallor Sweat yet is much disturbed by turning;		
	Nausea the nerves in the back of head get		
	cursed up.		
	CALORIC		
Douche RIGHT			
After 4 min. sec.	Nystagmus	1 to L	T to	about
Amplitude occasional movement	Motion sensing			5%
Total: min. sec.	Past-pointing			
	Pallor Sweat			
	Nausea			function
	Falling; general swaying			
Head Back →				
Amplitude bare response	Duration:	2 to L	2 to R	
Douche LEFT				
After 1 min. 35 sec.	Nystagmus practically no 1 to L		1 to L	
Amplitude occasional	Motion sensing only from horizontal canal			about
	Past-pointing very little			20%
Total: 4 min. sec. then quit and occasionally slight movement	Pallor none Sweat none			
	Nausea none			function
Head Back ←				
Amplitude good	Duration: 1/4 normal	¼ to L	3 to L	

taneous past pointing normal. After turning, nystagmus was normal in 29 per cent and subnormal in 63 per cent, and motion sensing was normal in 20 per cent and subnormal in 56 per cent. After douching, the responses from the vertical canals were normal in 33 per cent and subnormal in 56 per cent; responses from the horizontal canals normal in 29 per cent and subnormal in 67 per cent; motion sensing normal in 25 per cent and subnormal in 55 per cent; constitutional responses normal in 20 per cent, absent in 34 per cent, and subnormal in 46 per cent; past pointing normal in 38 per cent and subnormal in 46 per cent. In this group, therefore, normal and subnormal responses predominated (Chart 9).

Skull fracture was absent in 67 per cent of these patients, while 17 per cent had fractures of the vault and base (Chart 10). The remaining group had depressed fractures, vault fractures and basal fractures. True vertigo was absent, in 51 per cent postural dizziness

13. ANALYSIS OF CASES WITH CENTRAL LESIONS 26 CASES

	CASES	PERCENT
I. SPONTANEOUS VERTICAL NYSTAGMUS		
Present	1	4
Absent	24	92
Not given	1	4
II. SPONTANEOUS PAST-POINTING		
Normal	23	96
Exaggerated	1	4
III. TURNING		
Nystagmus		
None	1	7
Normal	6	24
Exaggerated	4	15
Subnormal	15	58
Perverted	0	0
Not given	0	0
Motion Sensing		
Normal	8	31
Exaggerated	4	16
Subnormal	10	39
None	1	3
Not given	3	11
IV. CALORIC		
Vertical Canals		
Normal	8	30
Exaggerated	1	4
Subnormal	14	54
None	1	4
Perverted	1	4
Not given	1	4
Horizontal Canals		
Normal	2	7
Exaggerated	0	0
Subnormal	21	81
None	0	0
Perverted	2	7
Not given	1	4
Motion Sensing		
Normal	5	20
Exaggerated	0	0
Subnormal	15	58
None	3	11
Not given	3	11
Constitutional Responses		
Normal	1	4
Absent	17	65
Exaggerated	1	4
Subnormal	6	23
Not given	1	4
Past-pointing		
Normal	10	39
Exaggerated	4	15
Subnormal	11	42
Not given	1	4

was present, general dizziness in 25 per cent, combined dizziness in 8 per cent and dizziness not present in 16 per cent. Postural dizziness occurred most frequently, but all forms were present except true vertigo. Eighty-four per cent of these patients were unconscious from a few moments to twelve hours, while 16 per cent of them did not lose consciousness.

CASE 2 (End Organ Lesion).—J. S., male, age 63 years, on July 23, 1929, jumped from a burning auto truck, striking his head; he was rendered unconscious for a half hour. He was hospitalized for a period of one month. Following the injury he immediately complained of dizziness, together with deafness and a ringing in his right ear. His hearing slowly improved and the ringing subsided. The dizziness continued, occurring in attacks which each lasted a few seconds; such attacks were brought on by rapidly turning his head to one side or the other, or up and down, by stooping, by riding in street cars or elevators, walking in traffic and by physical exertion. Occasionally, when the dizziness was most severe, he staggered.

Physical examination revealed an ambulatory man; temperature, 98.6; blood pressure, 130/80; height 5 ft. 7 in.; weight, 125 pounds.

14. SYMPTOMATOLOGY IN CENTRAL LESIONS 26 CASES

	CASES	PERCENT
I. SKULL FRACTURE		
1. Absent	5	20
2. Depressed	4	15
3. Vault	4	15
4. Base	8	30
5. Vault and Base	5	20
II. DIZZINESS		
1. Vertigo	0	0
2. Postural	12	46
3. General	4	15
4. Combined	0	0
5. Absent	10	39
III. SEVERITY OF INJURY		
1. Not unconscious	2	7
2. Momentarily	1	3
3. Unconscious period		
a. 1/2 hour	6	24
b. 1/2 to 2 hours	4	16
c. 2 to 12 hours	1	3
d. 12 to 48 hours	7	27
e. 48 hours to 1 month	5	20

15. Measurements with Audio-Amplifier.

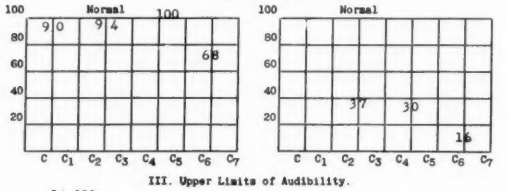
RIGHT EAR LEFT EAR.

I. Amplifier.

Voice, confused ____, distinct fairly Voice, confused slight, distinct ____.

Whisper, percentage hearing 100% . Whisper, percentage hearing none .

II. Percentage Hearing for Tones.



III. Upper Limits of Audibility.

14,000 d.v. ____ d.v.

IV. Bone Conduction.

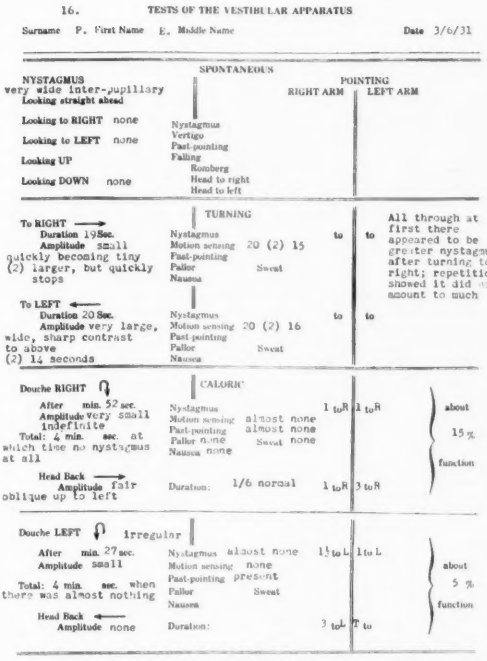
128 85 , 256 94 , 512 109 . 128 112 , 256 112 , 512 118 .

Neurologic examination showed no abnormalities.

The vestibular and auditory tests were performed by Isaac Jones. The auditory chart (Chart 11) demonstrated that the amplified voice was very confusing to both ears, and patient did not hear the whispered voice. In the right ear, hearing was limited to the lower and middle registers, but he did not hear above 3,000. The upper limit in the left ear was 8,000. The average hearing in the right ear was 40 per cent, in the left, 45 per cent. Vestibular examination showed subnormal responses upon turning, 40 per cent. Caloric responses demonstrated 5 per cent function in the right ear and 20 per cent function in the left. This indicated a marked impairment of both internal ears in the vestibular and cochlear portions (Chart 12).

CENTRAL LESIONS

Twenty-six patients (40 per cent) had central lesions; ten of this group, in addition, had end organ lesions Spontaneous vertical



16. TESTS OF THE VESTIBULAR APPARATUS

Surname P. First Name E. Middle Name Date 3/6/31

SPONTANEOUS

NYSTAGMUS
very wide inter-pupillary
Looking straight ahead

Looking to RIGHT none
Looking to LEFT none
Looking UP
Looking DOWN none

Nystagmus
Vertigo
Past-pointing
Falling
Romberg
Head to right
Head to left

POINTING
RIGHT ARM | LEFT ARM

TURNING

To RIGHT →
Duration 19 Sec.
Amplitude small quickly becoming tiny (2) larger, but quickly stops

Nystagmus
Motion sensing 20 (2) 15
Past-pointing
Pallor Sweat
Nausea

to to

All through at first there appeared to be greater nystagmus after turning to right; repetition showed it did not amount to much

To LEFT ←
Duration 20 Sec.
Amplitude very large, wide, sharp contrast to above (2) 14 seconds

Nystagmus
Motion sensing 20 (2) 16
Past-pointing
Pallor Sweat
Nausea

to to

CALORIC

Douche RIGHT
After min. 52 sec.
Amplitude very small indefinite
Total: 4 min. sec. at which time no nystagmus at all

Nystagmus 1 to R 1 to R
Motion sensing almost none
Past-pointing almost none
Pallor none Sweat none
Nausea none

about 15 % function

Head Back →
Amplitude fair oblique up to left

Duration: 1/6 normal 1 to R 3 to R

Douche LEFT irregular
After min. 27 sec.
Amplitude small
Total: 4 min. sec. when there was almost nothing

Nystagmus almost none 1½ to L 1 to L
Motion sensing none
Past-pointing present
Pallor Sweat
Nausea

about 5 % function

Head Back ←
Amplitude none

Duration: 3 to L T to

nystagmus was absent in 92 per cent of the cases and present in four. Spontaneous past pointing was normal in 96 per cent and exaggerated in 4 per cent. After turning, nystagmus was normal in 24 per cent and subnormal in 58 per cent; motion sensing normal in 31 per cent and subnormal in 39 per cent. After douching, the responses from the vertical canals were normal in 30 per cent and subnormal in 54 per cent; from the horizontal canals, nystagmus was normal in 7 per cent and subnormal in 81 per cent; motion sensing was normal in 20 per cent and subnormal in 58 per cent. Constitutional responses were absent in 65 per cent and subnormal in 23 per cent. Past pointing, after douching, was normal in 39 per cent and subnormal in 42 per cent. Here again, the normal and subnormal responses predominated, with constitutional responses absent and subnormal in the majority of the cases (Chart 13).

Twenty per cent of these patients did not exhibit a fractured skull, whereas 80 per cent had received fractures of different varieties (Chart 14). Postural dizziness predominated here, occurring in 46

17. TYPE OF SKULL INJURY IN RELATION TO VESTIBULAR LESIONS

	CASES	PERCENT
I. NONE		
1. Normal	10	36
2. End Organ	13	47
3. Central	1	3
4. End organ & Central	4	14
II. DEPRESSED		
1. Normal	2	25
2. End Organ	2	25
3. Central	4	50
4. End Organ & Central	0	0
III. VAULT		
1. Normal	1	11
2. End Organ	2	22
3. Central	3	33
4. End Organ & Central	3	33
IV. BASE		
1. Normal	1	6
2. End Organ	5	34
3. Central	5	34
4. End Organ & Central	4	26
V. VAULT & BASE		
1. Normal	2	33
2. End Organ	2	33
3. Central	2	33
4. End Organ & Central	0	0

per cent of the cases; yet 39 per cent did not complain of dizziness in any form. Of this group, 93 per cent were unconscious for periods varying from a few moments to several hours, a suggestion, to a degree, of severe injury.

Case 3 (Central Lesion).—E. P., male, aged 29 years, on May 19, 1929, was struck on the head in a hold-up. He remained unconscious for seven days. There was bleeding from the left ear; it was necessary to operate for a middle meningeal hemorrhage. Upon regaining consciousness, patient complained of both loss of hearing and of ringing in his left ear, headaches, blurred vision and insomnia. Returning to his occupation as a structural steel worker, he carried on satisfactorily.

On February 3, 1930, he felt queer, making many mistakes at his work. Early the following morning his left arm felt numb, and he noticed inability to lace his shoes. The left side of his face, the left arm and left leg suddenly began to twitch and he became unconscious. His father, who was observing him, stated the left side of patient's body convulsed violently. He did not regain consciousness for four hours, and thereafter, in spite of luminal therapy, repeated convulsions occurred throughout the following two years. Encephalogram was performed September, 1929, and since that time the patient has been comparatively free from convulsions, having only one during the past two years.

Physical examination revealed an ambulatory male; blood pressure, 130/70; weight, 135 pounds; height, 5 ft. $6\frac{1}{2}$ in. Scars were present in the left tympanic membrane approximately in front of the umbo and in the posterior inferior quadrant. Neurologic examination divulged a large bone-flap scar in the right temporal parietal region. The pupils reacted sluggishly to light. X-ray of the skull indicated a fracture line extending from the glenoid fossa, upward and backward, to the squamos portion of the temporal bone and half way across the parietal bone. A large opening (6 x 9 cm.) occurred in the right parietal frontal area, and a bone flap, in which areas of absorption were present, covered the upper two-thirds.

18. TYPE OF DIZZINESS IN RELATION TO VESTIBULAR LESION

	CASES	PERCENT
I. NO DIZZINESS		
End organ lesions	6	30
Central lesions	7	35
Normal vestibular	5	25
End organ and central	2	10
II. POSTURAL DIZZINESS		
End organ lesions	12	37
Central lesions	9	27
Normal vestibular	5	16
End organ and central	5	16
III. GENERAL DIZZINESS		
End organ lesions	6	40
Central lesions	0	0
Normal vestibular	6	40
End organ and central	3	20
IV. COMBINED DIZZINESS		
End organ lesions	3	100
Central lesions	0	0
Normal vestibular	0	0
End organ and central	0	0

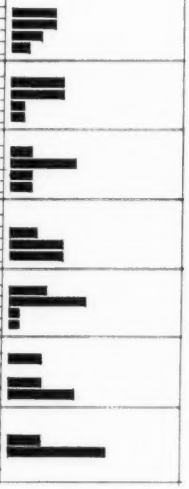
Auditory and vestibular tests were performed by Isaac Jones. The auditory chart (Chart 15) demonstrated normal hearing of the right ear, and lack of function of the left cochlea, although the bone conduction was normal for lower frequencies. The vestibular examination showed a 5 per cent function in the left ear; such an absence was caused by the fracture, which extended through the internal ear. The right vestibular function was 15 per cent. The presence of spontaneous vertical nystagmus upwards, and the existence of oblique nystagmus upward and to the left, after douching the right horizontal canal, would suggest an involvement of the vestibular pathways subtentorially in the brain stem (Chart 16). Encephalogram (Fig. 1) displayed extreme cortical atrophy and moderately dilated ventricles; Fig. 2 showed a definite pull of the posterior horn toward the side of brain damage.

To determine whether any particular types of skull injury were associated with specific vestibular responses, Chart 19 was constructed. Normal vestibular responses were present in patients with all types of fractures. Those patients without fractures exhibited all types of vestibular response. In those patients without skull fracture, however, only 3 per cent had central lesions, while in those with basal fractures 67 per cent had central lesions. Central brain damage, therefore, was present in the more severely injured patients.

Many investigators are of the opinion that dizziness caused by head injuries is secondary to lesions in the vestibular mechanism. Some have felt the neuro-otologic tests could indicate the presence or absence of dizziness, which would thereby differentiate the malingerer from the actually injured person. They felt, further, the duration of dizziness could be prognosticated by such tests. These facts are definitely refuted in Chart 20. Seventeen, or 25 per cent, of the patients did not complain of dizziness, yet fourteen, or 83 per cent, of this latter group had abnormal vestibular responses. Further,

19 PERIOD OF UNCONSCIOUSNESS IN RELATION TO VESTIBULAR LESIONS

	CASES	PERCENT
I. NOT UNCONSCIOUS		
1. Normal vestibular	3	33
2. End organ	3	33
3. Central	2	22
4. End organ & central	1	11
II. MOMENTARY		
1. Normal vestibular	4	40
2. End organ	4	40
3. Central	1	10
4. End organ & central	1	10
III. ONE-HALF HOUR		
1. Normal vestibular	2	16
2. End organ	6	50
3. Central	2	16
4. End organ & central	2	16
IV. 1/2 to 2 HOURS		
1. Normal vestibular	0	0
2. End organ	1	20
3. Central	2	40
4. End organ & central	2	40
V. 2 to 12 HOURS		
1. Normal vestibular	4	28
2. End organ	8	58
3. Central	1	7
4. End organ & central	1	7
VI. 12 to 48 HOURS		
1. Normal vestibular	2	25
2. End organ	0	0
3. Central	2	25
4. End organ & central	4	50
VII. 1 WEEK to 1 MONTH		
1. Normal vestibular	0	0
2. End organ	2	25
3. Central	6	75
4. End organ & central	0	0



whereas patients complaining of dizziness and who were examined from time to time, showed no essential difference in their vestibular abnormalities, still at a later date their dizziness subsided. As will be later demonstrated, both the encephalograms and vestibular tests performed on four patients showed evidence of brain damage; two of these patients, despite the existence of such pathology, reported no dizziness. That dizziness is not a prerequisite of vestibular tract pathology is further substantiated by the numerous cases of neurologic diseases and tumors of the brain which, with normal vestibular response, still complained of dizziness. This clearly demonstrates that lesions of the central or peripheral pathways may exist, yet dizziness be entirely absent. The various types of dizziness—such as postural, general and combined—may occur with all types of vestibular abnormalities; the abnormalities, therefore, found upon vestibular examination can in no manner determine the type of dizziness from which the patient complains; furthermore, in view of the abnormalities which continue to persist in spite of the subsidence of dizziness, the duration of such dizziness can in no way be prognosticated.

The cases studied have been further analyzed to determine whether the period of unconsciousness is in any manner related to the type of vestibular response. A review of Chart 21 divulges the ab-

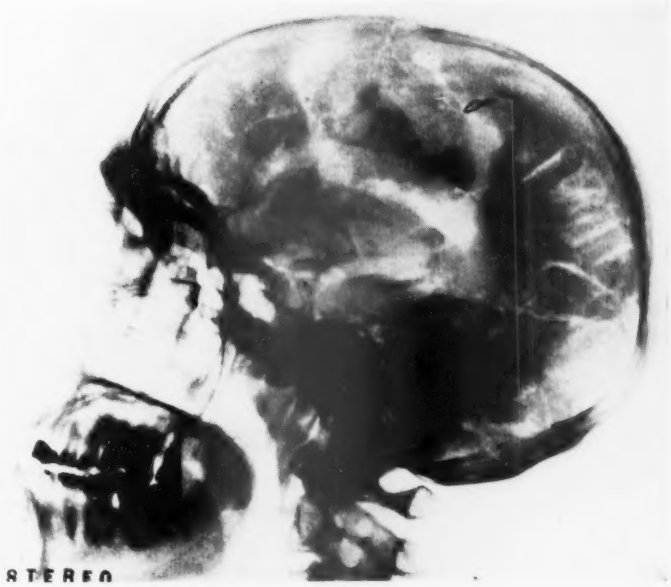


Fig. 1. Marked cortical atrophy.

sence of such a relationship and shows that every form of vestibular response is associated with all periods of unconsciousness.

Auditory examinations were performed by Isaac Jones with an audiometer in a soundproof room. One patient revealed a complete loss of hearing in the right ear and 3 per cent loss in the left. In three cases there was 30 per cent hearing in the left ear. In three cases there was 40 per cent hearing in the right, and 40 per cent in the left. In two cases there was 50 per cent hearing in the right ear, and in four cases 50 per cent in the left. Sixty per cent hearing occurred in the right ear for six cases, in the left ear for eight cases. Seventy per cent hearing registered for nine cases in the right ear and six cases in the left ear; 80 per cent hearing was disclosed for nine cases in the right ear and six cases in the left; 90 per cent hearing for ten cases in the right ear and ten in the left; 100 per cent hearing for eleven cases in the right ear and nine in the left. The hearing defects were below 60 per cent in eleven cases, and the remaining forty-five above 50 per cent.

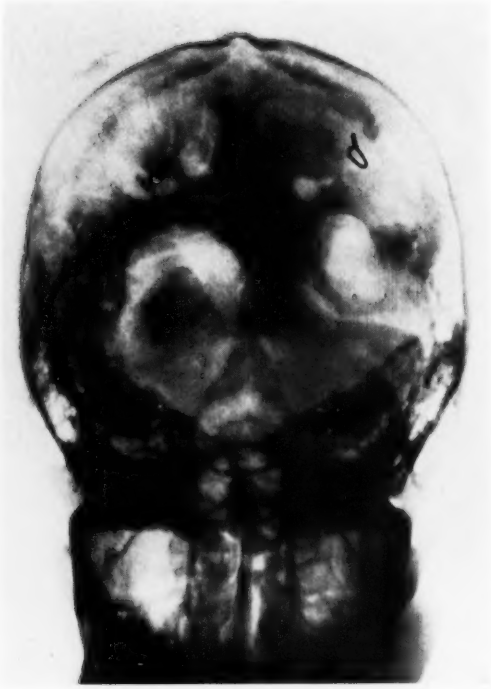


Fig. 2. Marked cortical atrophy, ventricular dilatation and ventricular shift.

Bleeding from the ear was present in fourteen cases, seven of which had tinnitus, and five a hearing loss of below 60 per cent. Tinnitus occurred in seventeen cases; in five of these the range of hearing was between 70 and 100 per cent. All of the cases had cochlea damage, except for two with middle ear involvement. The patients with hemorrhage through the tympanic membrane may have normal hearing or a loss of hearing; they may have tinnitus or may not have tinnitus; tinnitus may occur with normal hearing or with diminished hearing.

Encephalograms were performed on four of these patients, all of whom had central lesions upon vestibular examination; two only complained of dizziness. Encephalograms revealed abnormalities in the entire group, such as cortical atrophy, dilated and irregular and unequal ventricles and a ventricular shift. In these four patients the

encephalogram and the vestibular test corroborated the presence of brain pathology, yet dizziness occurred in only two of this group (Figs. 1 and 2).

SUMMARY

The vestibular tests are of no value in determining either the type of dizziness or the presence or absence of dizziness encountered in these patients, nor by them can the duration of dizziness be prognosticated. They do, however, in certain cases, reveal the presence of pathology within the brain and thus corroborate, to a degree, the truth of the patient's story in regard to a head injury. In this manner they are indirectly of value in eliminating the malingerer. It is quite evident that the common types of dizziness associated with head injuries are not dependent upon definite vestibular lesions; instead, it is likely that they are due to an entirely different mechanism, possibly transient cerebral vasomotor disturbances.

Encephalograms and vestibular tests, performed on four patients, both demonstrated central brain pathology. In two of these patients dizziness was entirely absent.

In a series of 325 cases of head injury, which were followed for a period of one to eight years, 67 per cent of the patients complained of headaches and 60 per cent of dizziness. Vestibular tests were performed upon sixty-six of the whole group. Subjective symptoms were present in 80 per cent; objective neurologic signs in 21 per cent, and vestibular abnormalities were evident in 76 per cent of these. Objective signs were thus demonstrated more frequently by vestibular examination than by clinical neurology.

Of skull fractures present in 56 per cent of this group, 22 per cent were basal, 13 per cent vault, 12 per cent depressed and 9 per cent vault and basal. Eighty-seven per cent of these patients were unconscious, of whom 15 per cent were momentarily so; 18 per cent, from a few minutes to a half hour; 7 per cent, from one-half hour to two hours; 23 per cent, two to twelve hours; 12 per cent, twelve to forty-eight hours, and 12 per cent, forty-eight hours to one month.

Though no distinct vestibular complex was associated with head injuries, there were certain predominate findings. Spontaneous responses were normal; responses, after turning, were normal or subnormal; caloric rseponses were normal or subnormal; and constitutional responses, after douching, were normal, absent or subnormal. Normal vestibular responses occurred in 24 per cent, end organ lesions in 36 per cent and central lesions in 40 per cent.

Normal vestibular responses may indicate either an undamaged brain or a brain evidencing pathology which has not involved the vestibular pathways. End organ lesions naturally signify peripheral involvement of the vestibular fibers, while central lesions indicate pathology either directly present or adjacent to the central vestibular pathways.

Dizziness has been arbitrarily divided into true vertigo, postural dizziness, general dizziness and combined dizziness. In this series true vertigo was absent; postural dizziness ocurred in 40 per cent; generalized dizziness in 22 per cent; combined dizziness in 4 per cent, while 25 per cent did not complain of dizziness. Exceedingly difficult to understand, however, is the absence of a history of true vertigo in head injuries, in spite of the presence of peripheral and central vestibular damage.

Head injuries may damage the cochlea so as to cause various degrees of deafness. Hemorrhage of the ear with rupture of the tympanic membrane may occur with perfectly normal or diminished hearing; tinnitus, too, may be present or absent.

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XXXVI

SOME FACTORS INFLUENCING MORTALITY IN CASES OF FOREIGN BODY OF THE LOWER AIR AND FOOD PASSAGES*

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Among the many good and wise rules laid down by that master endoscopist, Chevalier Jackson, are the two following aphorisms:

1. "Cry over spilled milk enough to memorize how you spilled it."

2. "Let your mistakes worry you enough to prevent repetition."

It is in the spirit of the above injunction that after an endoscopic experience of some ten years and approximately 300 foreign body cases, the mortality arising during this period will be reviewed. Attention will be focused on those factors which it would seem had a direct influence upon the unfavorable outcome in each of these cases.

A total of fifteen cases have come under observation during this time. Of these, five were not operated upon, for reasons that will be set forth in the individual case reports. Ten cases were operated upon. Of these, the foreign body was removed in six cases. In the case of one very small infant with a very large, open safety pin, the pin escaped downward into the stomach; in another it was purposely guided into the stomach (Case 10). In two cases, the foreign body was not recovered at the first operation, and the patient expired before another attempt was thought advisable. In two cases, a foreign body was removed, but another one remained hidden deep in a secondary bronchus.

From a rather close study of the above, we hope to point out that the following factors play a predominant part in the mortality arising from foreign bodies in the upper air and food passages.

1. Faulty and delayed diagnosis.
2. Improper manipulation.
    a. finger.
    b. bouginage.

*Presented as a Candidate's thesis for admission to the American Laryngological, Rhinological and Otological Society.

3. Improper preparation of the patient.

4. Extreme youth of the patient.

5. Anesthetic.

6. Lack of competent, specially trained personnel for assisting at the operation and lack of a proper instrumentarium.

7. The proper judgment in the use of tracheotomy.

8. Perforation of the esophagus.

9. Postoperative care.

REPORT OF CASES

CASE 1.—C. S., a white female, aged 27, applied at the out-patient department of the Buffalo City Hospital on December 28, 1935.

The patient stated that the previous day, without apparent cause, she had coughed up a cupful of dark, red blood; and a small amount of bright blood just before coming to the hospital. Her past history was of no significance, except that she had had "colds on the chest" at frequent intervals in the past six months. The patient was not losing weight, had had no night sweats or known tuberculosis contacts.

Because of the recent bleeding, she was admitted to the medical wards of the hospital, and put to bed. The following day, she bled a sputum cupful of bright red blood. On December 29, there was a moderate amount of hemorrhage, and the patient became very weak. The heart tones were poor, and the temperature rapidly mounted from 98.8 to 106. The patient expired.

During her short stay in the hospital, a thorough clinical examination was impossible because of the bleeding. X-ray taken in bed, showed a fine infiltration of the inner portion of the right base. This was considered the result of hemorrhage. The rest of the lung fields were clear. The heart and aorta were within normal limits. Both diaphragms were normal in position and contour.

The following notes are taken from the report of the autopsy:

"Lungs.—The right upper, right middle, and left upper and lower lobes are grey-pink in color, and crepitate normally throughout. The right lower lobe is atelectatic, deep cyanotic red in color, and does not crepitate at all, except in the upper anterior portion. The hyperlateral bronchus is completely filled with a fresh, soft, unorganized, deep red blood clot. The bronchial mucous membranes are stained deep red. Ten cm. from the tracheal bifurcation in the right lower main bronchus, at its division into several smaller collaterals, there is a firm, granular, rough, pea-sized foreign body, measuring 5 by 5 mm. After cleaning, it is white in color, and grossly resembles a quartz pebble or piece of shell. The secondary bronchial mucous membrane is dirty red in color, with a shallow ulceration at the site of the foreign body. The contiguous membrane is coarsely granular. No other bleeding point is discovered."

CASE 2.—S. K., a white male, aged 23, was admitted to the Buffalo City Hospital, December 11, 1933.

The patient stated that his tonsils had been removed five weeks previously, since which time there had been occasional bleeding from the nose and throat.

Three days before entering the hospital, he caught cold, and after returning from work that night, was seized with chills, numbness of the extremities, and general prostration.

On the day of admission, he was seized with a coughing spell, and coughed up a large quantity of blood. The family physician called an ambulance and sent him to the hospital.

The patient stated that for several years he had caught cold easily, and had had what he termed "bronchitis" for the past nine years.

The temperature upon admission was 102, the pulse, 120, and the respirations 22 per minute. The patient was first seen in the nose and throat service, where the only positive finding at the time was a blood clot in the left nostril. A general physical examination was negative. The blood pressure was 120/80.

The case was diagnosed as grippe, and transferred to the medical department at once.

Three days after entering the hospital, the patient began coughing up red, frothy blood, which continued at intervals during his stay in the hospital. Further examination showed some immobility of the left chest, and occasional rales. The temperature was irregular, ranging from normal to 104.

The urine showed a 3 plus albumen on one occasion, and at other times was negative. The blood urea was 14, and the blood sugar, 206. The blood count on December 12 was 70 per cent hemoglobin, reds 4,330,000. Whites were 5,360, of which polymorphonuclears showed 32 per cent segmented, 16 per cent stab forms, 43 per cent lymphocytes and 9 per cent mononuclears.

An x-ray on December 13 showed the lung fields clear. There was no evidence of any pneumonic process. The hilar regions showed a slight increase in size and density. The heart and aorta were within normal limits. December 16, an x-ray showed some fine infiltration in the left base, probably the result of hemorrhage. The remainder of the lung fields were clear.

A physical examination done on this date by the chief consultant of the chest service showed very little evidence of intrapulmonary disease. He considered the hemoptysis due to a very small intrapulmonary lesion, and advised pneumothorax. The same day, 600 cc. of air was injected into the pleural cavity.

On December 17 the patient bled 200 cc. of blood, after 500 cc. of air had been given. He bled very little after this for the next few days. On December 30, 450 cc. of air was injected. On January 1 a severe coughing spell took place, and a sharp bone, measuring 1 inch in length, was raised. This was accompanied by hemorrhage.

I was called to see the patient at this time, but insofar as the foreign body had evidently removed itself and the patient was still bleeding, we advised against any manipulation.

Although the patient was transfused and given every possible care, he continued bleeding, and expired four days later. After coughing out the bone, the patient gave the following history:

Nine years previously he had swallowed a chicken bone. He felt it in his throat at the time, but was unable to extract it with his finger. He went to a

physician who x-rayed him and said that there was no bone present. Since that time the patient had had what he described as "severe chest colds," accompanied by paroxysms of coughing, fever, and foul discharge. In between attacks, he was not bothered except by a mild, chronic cough, and an occasional pain over the left chest. He saw several doctors during this period, and at various times asked if the bone he swallowed could be causing the trouble. He was always assured that there was no bone present, and finally gave up mentioning it when he sought medical attention.

CASE 3.—S. K. O., a Polish laborer, aged 43, applied to the outpatient department of the Buffalo City Hospital, June 1, 1935.

In October, 1934, the patient had choked on a bone which went down the "wrong way." Shortly after that he began coughing. This cough was at first dry, and later productive of foul, yellow pus. He lost 27 pounds of weight in the six months previous to his application to the hospital, and on the date of admission was bringing up half a cupful of pus a day.

An examination in the outpatient department led to the diagnosis of pulmonary fibrosis, chronic bronchitis, with bronchiectasis, and possible foreign body of the lung.

X-rays taken at this time showed some fibrosis throughout the lungs, and an area in the right base which was suggestive of bronchiectasis. The heart was enlarged to the left; the aorta was normal. The diaphragm was normal in position and contour.

The examining physician referred this patient to the chest clinic, recommending bronchoscopic examination for foreign body. The history of the aspiration of a foreign body was lost sight of. Expectorant mixtures were given and the patient was told to report at two week intervals. He made no improvement, in fact, became worse.

On October 2, 1935, he returned to the hospital, reporting that he had coughed up a piece of bone one inch long, afterward raising blood flecked purulent sputum. This was preceded for about a week by pain in the right chest and night sweats. He was admitted to the medical service for physical examination, which showed a possible abscess in the right lower lobe.

An x-ray study of the chest at this time confirmed the diagnosis of a right lower lobe abscess. The left side of the chest and right upper lobe were clear. In the lower right lobe was a large abscess with fluid level. The film with the affected side up showed a marked thickening of the pleura along the entire axilary line, with a shift in the fluid level so that all the lower ribs were visualized. The heart was displaced to the left.

The patient's condition rapidly grew worse. Thoracotomy was considered, but his condition did not permit of surgery. He expired on October 20, 1935.

CASE 4.—S. G., a white male, aged three and one-half years, entered the Buffalo City Hospital on a Saturday night, June 26, 1931.

The history obtained upon admission was very meagre, due to the lack of an interpretor. The parents stated that the child had been sick for four weeks with cough and fever, had been getting worse the past week, and much worse the past twenty-four hours.

The temperature upon admission was 100, the pulse 144, and the respirations 48. Examination showed a very sick child, lying with head retracted, and breathing with much stridor. Auscultation showed an inspiratory rattle, and the breath sounds were markedly diminished in both lower lobes, which revealed a diminished resonance. The heart rate was moderately increased and toxic.

The next day, the following history was obtained. Four weeks previously the boy had been playing with a bead. Some time later he came into the house vomiting, and when this persisted intermittently for an hour, a physician was called. The child breathed with difficulty, and had a persistent cough, which was occasionally followed by emesis. He admitted that he had swallowed the bead, which was not recovered. Within a few days, the acute symptoms disappeared. The cough persisted, the child frequently expectorating pus for a period of three weeks, after which the cough was unproductive. He became somewhat better.

Four weeks after the onset of the illness, on the day of admission to the hospital, the patient was seized with chills and fever. He became quite ill, and the parents brought him to the hospital.

The day after admission, before the history had been obtained, several diagnoses were entertained. First, lung abscess; second, tuberculosis with meningitis, and third, poliomyelitis, bulbar type. Spinal puncture yielded a clear fluid under slight pressure. The right ear was infected, and a paracentesis was performed.

On Monday, the 29th, the patient was seen by the chief pediatrician who made the following observations: "Marked malnutrition. The right ear drum shows the effect of recent incision. The mouth is dry, with ulceration on both cheeks, and there is evidence of dental caries. The neck shows no rigidity. The chest is dull over the right side, anterior and posterior, and over the middle and lower lobes. There is some impairment over the right upper lobes, anteriorly and posteriorly. The breath sounds over the middle and lower lobes are diminished. Scattered ronchi and coarse rales are present in the right infraclavicular area. The heart sounds are louder to the right of the sternum, apparently due to a shift of the mediastinum. The heart rate is about 160, and the tones are fair." The rest of the examination was negative.

An x-ray was ordered and showed the presence of a large, oblong shadow, 2.8 by .9 cm., lying in the right bronchus, just below the bifurcation of the trachea. The lung fields were clear, but the right lung showed a spotty infiltration in the base, with a displacement of the heart and the trachea to the right. The diaphragm was not elevated.

The patient was referred for bronchoscopic removal of the foreign body, but expired before it could be done, soon after the x-ray was taken.

At postmortem examination removal of the chest plate revealed that both pleural spaces were clear. On the right side, the visceral pleura was somewhat thickened, and covered with a fibrinous exudate. Upon opening the trachea, a bead was found in the lumen. The trachea showed numerous small ulcers which were covered with a fibrinous purulent secretion. The right bronchus was filled with fibrinous and purulent flakes. After dissection of the right lung, all ramifications of the bronchi appeared markedly distended, pocket like, and filled with a greenish material. These pus cavitations extended outward to the visceral pleura, but did not perforate the latter. The left lung showed a few small areas of atelectasis, but otherwise revealed no pathological change.

CASE 5.—S. W. M., a white male, aged two years, was admitted to the Children's Hospital, July 31, 1931.

At 7:30 that morning, the maid had given the child a whole tablet of calcium compound on which he choked. He immediately became quite dyspneic, coughed, and gasped for breath. Unable to obtain a physician quickly, the parents brought the child to the hospital.

When admitted, he had stopped breathing, and the pulse was imperceptible. The artificial respiration, oxygen, and caffeine, were given without effect. The heart sounds were inaudible at all times, and the child made no attempt at breathing. I had been called, and arrived at the hospital just after the child entered—too late. The baby had expired within a very few minutes after aspiration of the foreign body, and before it reached the hospital.

CASE 6.—T. A., a white female, aged 68, entered the Mercy Hospital, August 14, 1935.

The patient gave a history of having swallowed a chicken bone three days previously, after which she had been able to swallow liquids only. She had had occasional pain in the epigastrium, and off and on a feeling of chilliness.

At the time of admission, her temperature was 103 (rectal), the pulse 100, and the respirations 28. The heart was rapid and the tone of poor quality. Auscultation revealed marked rales at both bases of the lungs, with a dull percussion note over the lower left lobe.

A barium gruel mixture passed normally to the level of the end of the sternum, where it narrowed down abruptly to a thin line. The roentgenologist's opinion was that the pathology so shown was not related to swallowing a bone.

The following day we were called to see the patient. At this time the temperature was 106.4, and the pulse 144. The respirations were 35 per minute. The patient was semicomatose and obviously critically ill. She had had several severe chills during the past 36 hours.

Our diagnosis was mediastinitis, due to a perforated esophagus. We advised esophagoscopy and removal of the foreign body, if present, but expressed the opinion that the patient's condition was practically hopeless.

Esophagoscopy was done under local anesthetic. At a level just below the aorta, a double chicken bone was discovered lying crosswise in the esophagus. One end of the bone had perforated the left wall, where the membranes were edematous, and exuding a serosanguinous fluid. The bone was easily removed.

No improvement resulted from the operation, and the patient died the third day postoperatively. No autopsy was obtained.

CASE 7.—R. R., a white male child, aged 9 months, was admitted to the Buffalo Children's Hospital, the evening of March 9, 1929.

The mother stated that two days previously the baby had swallowed a safety pin. She had turned the child upside down. He gagged, and afterward seemed to feel better. The following day there was no apparent pain, and the baby took his feeding well. Two days later, however, he seemed to have trouble swallowing, and a physician was called. He attempted to pass a stomach tube, but could not get it through, and sent the child to the hospital.

Examination showed a baby acutely ill, with a temperature of 101, a pulse of 150, and respirations at 38. A physical examination gave no positive findings.

X-ray showed a large, open safety pin lying transversely in the esophagus at the level of the aortic constriction. The point projected so far to the left that perforation seemed almost certain.

An esophagoscopy was performed without anesthetic, and the pin easily removed. The membranes around the point were slightly edematous.

Despite careful feeding and supportive treatment, the baby's condition grew worse. The temperature gradually rose, until the third day after admission it reached 105.2. The breathing became more labored and the baby expired.

The clinical diagnosis was mediastinitis. No autopsy was obtained.

CASE 8.—R. U., a white male, aged 18 months, was admitted to the Buffalo City Hospital, August 22, 1931.

A few hours before admission to the hospital, the child had swallowed a religious medal. The mother saw the baby swallow it, and was able to see the medal in the back of the throat. She attempted to remove it with her finger, but only succeeded in pushing it farther down, until she could no longer feel it. The baby was taken to the family physician, who sent him to the Buffalo City Hospital.

Examination showed a well-developed baby of stated age, with no abnormalities. The temperature, pulse and respirations were normal.

Fluoroscopic examination showed a large, square, metal medallion, with eight sharp, diverging projections at the corners and sides. This was lying in the upper esophagus.

At 11 p. m. an esophagoscopy, without anesthesia, showed the foreign body lying just below the cricopharyngeus muscle. It was tightly impacted, and resisted all attempts at removal. Every effort to rotate the foreign body was futile, since the sharp hooks at the corner always caught in the esophageal wall.

The day after admission the temperature mounted to 102.3, and the pulse was 150. Subcutaneous emphysema of the neck appeared, and the baby developed considerable dyspnea.

On the 24th, x-ray showed the foreign body lying to the posterior and right of the esophagus. There was also a complete collapse of the right lung due to pneumothorax.

Clinical findings corroborated these findings. The patient expired on October 25, three days after admission.

CASE 9.—L. B., a white male, 15 months old, was admitted to the Children's Hospital, October 12, 1930.

While changing the baby's diaper, on the day of admission, the mother missed a safety pin. About 45 minutes later, the baby began to choke, and breathe with difficulty.

Examination a few hours later showed a well nourished, well developed baby. He gave an occasional cough, but the physical examination was entirely negative.

An x-ray was taken and showed a large, open safety pin with the point directed upward in the middle third of the esophagus.

An esophagoscopy was performed without anesthetic. While attempting to free the point of the pin from the esophageal mucosa, the foreign body started slipping down the esophagus, entering the stomach before it could be removed.

The child remained in the hospital under observation. Fluoroscopic examination three days later, October 15, and also on the 17th, and 25th, showed the pin in the stomach.

Against our advice, the abdomen was opened, for the purpose of removing the pin from the stomach. The operation was unsuccessful, and the postoperative condition rather poor. For the next four days, the temperature ranged from 101 to 102.8, and then gradually fell to normal.

The day following the operation, October 28, a flat film was taken, and showed an open safety pin high up in the abdomen, apparently in the same position as previously. It seemed to be in the stomach, but the roentgenologist stated that it could be in the third portion of the duodenum.

Two days after this, October 30, a colon injection was given. The pin was in the same plane as the transverse colon, but appeared of greater size than the calibre of the organ. Palpation of the transverse colon did not move the foreign body, for it lay posterior to, and therefore in the third portion of, the duodenum.

On November 7, another x-ray examination was made, and the following report given:

"Barium meal has been administered and shows that the pin is not in the stomach. It is a trifle lower than the greater curvature, posterior to the stomach."

On January 26 an exploratory laparotomy was performed, and an open safety pin removed from the third portion of the duodenum. The child left the operating room in a fair condition, and was given 500 cc. of saline subcutaneously.

The following day the temperature went up to 102.4, and the pulse rose to 160. The child was given oxygen and 400 cc. of saline subcutaneously. He had a very poor night, and on the 28th the temperature mounted to 106, and the child expired.

The following notes are taken from the report of the autopsy:

"A well nourished male child, 87 cm. long. In the abdominal wall there is a laparotomy wound in the epigastrium. It is about 13 cm. long, and is closed with sutures. The margin of the fresh wound also shows scars resulting from laparotomy in the same place a few months ago. A few loops of the middle portion of the small intestine are attached to each other by fibrin. The wall of the intestine here shows a few fresh hemorrhages. The anterior portion of the stomach, along the greater curvature, is attached to the omentum magnum. The omentum is very much reduced in size, being about ½ cm. wide and .5 cm. thick. The omentum is adherent to the anterior wall of the abdomen."

"There is a second small area about 8 cm. long, in the middle portion of the jejunum, with a hemorrhage in the wall, and a slight fibrous thickening in the corresponding mesentery is observed. The beginning portion of the jejunum shows a fresh single suture, without any reaction. There is in general no erosion of the intestine; the serosa is smooth and glistening. There is only a slight amount of clear, serous fluid, and no free exudate. The intestine after opening shows a few small pin point hemorrhages in the lower colon and the rectum. Otherwise the

jejunum is almost entirely collapsed, and only the ileum is slightly distended. The duodenum is more markedly distended."

"The liver shows a diffuse, fatty degeneration, and is also apparently anemic. The gall bladder and the cystic duct are not present; congenital aplasia. The ductus choledochus and the hepatic duct are normal. The stomach mucosa reveals many pin point hemorrhages, and the stomach contains hemorrhagic mucous masses. The spleen is not remarkable. The kidneys show only distinct markings of the veins in the capsule; otherwise there is nothing remarkable. The adrenals and bladder are not remarkable. The lungs show atelectatic areas in the apex of the left lower lobe, and especially in the apices of the right lower and upper lobes. The lower lobe shows hemorrhages in the parenchyma and in the bronchioli, along with aspirated mucous from the stomach. Especially in this part, are the very small, pin point hemorrhages visible. Compressing the parenchyma, there is in all lobes, especially the lower ones, a marked serous secretion in the alveoli, but no pus is seen. The left upper and right middle lobe, along with the lower anterior part of the right upper lobe, show distinct emphysema in the marginal parts, also revealing distinct pin point hemorrhages in almost regular distribution. The right and left ventricles show a very slight dilatation, and some anemia. Dissection was made only by abdominal incision, and the brain and organs of the neck were not dissected."

CASE 10.—N. P., a white female, aged 8 years, entered the Buffalo Children's Hospital, July 15, 1936.

The mother stated that six weeks previously the child had developed an ordinary headache, with nasal discharge and cough, but no fever. She had fully recovered from this, but had remained listless and more or less apathetic, eating poorly. Five days before admission to the hospital the patient became acutely ill. She developed a fever, cried a great deal, breathed with difficulty, and grunted upon respiration. The family physician diagnosed bronchitis. There was no improvement after three days, and he sent the patient to the hospital with a diagnosis of pneumonia.

Examination showed an acute otitis media in both ears. There were signs of early bronchopneumonia, along with a small area of early consolidation of the left apex. The heart sounds were normal. The temperature upon admission was 104.6, the pulse 150, and the respirations 40.

A bilateral paracentesis was performed, after which the temperature dropped at once to subnormal, but returned again in a few hours to 104.

The roentgenologist made the following report after an x-ray of the chest.

"Lungs are clear. The heart shadow, especially on the left side, is abnormally large. The heart appears dilated, although the angles are clear cut. There is an open safety pin in the lower esophagus, with the point directed upwards."

Examination showed a normal urine. The blood picture revealed 24,000 white blood cells with 30 polymorphonuclears and 53 lymphocytes. The hemoglobin was 66 per cent.

On July 16, esophagoscopy was performed under a gas-oxygen-ether anesthetic. A large safety pin, sticking in the left wall of the lower esophagus, was found. The mucosa at this point was slightly edematous. Feeling that the pin had perforated, and not wishing to disturb possible barriers of infection, we carefully disengaged the pin and guided it into the stomach.

During the next eight days the patient had an irregular temperature, ranging from normal to 104.8. The pulse was around 130, and the respirations remained at about 40 per minute. On July 26 the temperature fell to normal, and for the next six days remained there, although the pulse still continued at about 130, and the respirations were very rapid.

On July 22 the patient passed the safety pin. The pediatrician made the following note:

"The child is eating poorly, but making a slight steady gain in weight, and looks slightly better. The heart sounds come through clearly, as they have right along, and therefore seem to rule out pericarditis as the cause of the chest shadow."

There had been some difference of opinion as to the cause of the large mediastinal shadow, some clinicians feeling that it was caused by a localized mediastinal infection, and others feeling that the shadow was entirely due to the heart. The roentgenologist made several examinations and called attention to the fact that films taken in the lateral position showed the posterior mediastinum to be clear.

On August 1 the child again had an irregular fever, which after three days dropped to normal. The pulse remained around 130. Clinicians found a few rales in the chest. The respirations remained frequent, occasionally accompanied by grunting, and cyanosis frequently appeared. The child's condition rapidly grew worse. On August 8 severe dyspnea and increased cyanosis were present. The heart tones were poor and irregular. The patient again had a fever.

On October 9 the child expired.

An autopsy was performed. Among the significant findings, there was a greatly enlarged pericardial sac, filled with pus. The culture gave a pure growth of pneumococcus. There was no evidence whatsoever of mediastinitis. There was no pneumonia. On the left wall of the esophagus, adjacent to the pericardium, there was a small spot of discoloration where the pin point had evidently perforated, setting up an infection in the pericardium.

Case 11.—D. H. J., a white male, aged 13 months, entered the Buffalo City Hospital about 10 a. m., October 14, 1934.

According to the mother, the baby suddenly began choking the previous evening, while playing on the floor. She turned the child upside down, and a half a salted peanut fell from his mouth. As he drew his breath again, he choked slightly. The baby coughed a little before going to sleep, but rested well until 5 a. m., when he began to choke and cough. The mother called the family physician, who sent the child to the hospital.

Examination at the time of admission to the hospital showed a well nourished baby, who constantly cried, and occasionally coughed. His temperature was 100 (rectal), the pulse 130, and the respirations approximately 40 per minute. A physical examination was difficult to obtain because of the constant crying, but there were a few signs on the left side that made the examiner suspicious of a foreign body there.

At 11:45, the baby was brought to the operating room. He was placed on the table, and while the instruments were being inspected he suddenly stopped crying and grew very cyanotic. A laryngoscope was quickly passed, and a peanut, jammed up just below the vocal cords, was seen obstructing the trachea. This was immediately pushed down and the baby began to breathe again. A 4 mm.

bronchoscope was passed, and the peanut was quickly removed. The whole operation lasted about three minutes, and the child was immediately placed in an oxygen tent.

The patient rested well that afternoon and evening. His temperature rose to 102.2 (rectal). During the night, however, he became quite restless and an interne was called. He noted that the baby was breathing rather harshly and that there were a few moist rales in the chest, and ordered 1/12 gr. morphine.

Two hours later, the baby became quite dyspneic, and we were called. A racheotomy was indicated, and performed under local anesthesia.

The patient's respirations became more free at once, but the general condition did not improve. A pediatrician examined the baby later in the day, and observed that he was acutely ill. He noted that there was no change in the percussion note of the chest, but that auscultation showed many coarse rales in the right base. The heart was rapid, and the tone only fair quality. He diagnosed early pneumonia.

Despite all supportive treatment, the baby's condition rapidly declined. His temperature mounted to 106, the pulse to 160, and the respirations to above 40. He expired 40 hours postoperatively.

An autopsy was obtained, and the following observations were made.

"There is a moderate edema of the vocal cords and subglottic tissues. The mucous membrane of the trachea and bronchial tree is markedly reddened and roughened, with a thick yellowish brown, tenacious mucus adherent. This mucus and inflammatory exudate are present to such a marked degree that they plug some of the lower and larger bronchi. When this exudative material is removed from the mucous membrane, it leaves a rough surface which reveals many pin point areas of red discoloration and superficial ulceration."

"The right lower bronchus is especially involved, and some of the lower bronchi are entirely occluded by mucous plugs. The right lower lobe crepitates poorly, and shows definitely a lowered air content. The tracheobronchial lymph nodes are somewhat swollen. The anatomical diagnosis is first, acute exudative tracheitis, bronchitis and bronchiolitis; second, pulmonary atelectasis; third, asphyxia, and last, cardiac dilatation.

CASE 12.—R. L., a white female, aged 9, entered the Buffalo City Hospital at 10:15 p. m., December 12, 1931.

The mother stated that the child had choked on a shell while eating nuts a few hours previously. Since then, she had been coughing and breathing with difficulty.

Examination showed the child in very poor condition. Breathing was labored, and all the muscles of respiration were being used. There were bloody crusts around the nose, and the patient was coughing a bloody, frothy fluid. Cyanosis was so marked that after fluoroscopy she was kept in an oxygen tent while the operating staff was being assembled. Auscultation showed an area of jerky, suppressed sounds at the level of the fifth and sixth rib, over the sternum; there was a slight limitation of motion over the right side.

Fluoroscopy was negative except for a slight lag of the right diaphragm.

The child was brought to the operating room at 11:30 p. m. She had been receiving oxygen continuously through a mask, and its removal caused immediate and extreme cyanosis.

Upon attempting to pass a bronchoscope, the child struggled violently, and there was an immediate emesis of considerable vomitus, consisting almost entirely of coarsely chewed nut kernels. Bronchoscopy had to be deferred until this was thoroughly aspirated. Because of the violent struggles of the child, who was very large for her age, a light anesthetic of gas and oxygen was deemed advisable, and was administered by a skilled anesthetist. Due to the patient's poor general condition, the anesthesia was kept very light, but there was violent emesis of more ground nuts, each time bronchoscopy was started. After considerable difficulty, a 6 mm. bronchoscope was passed. Oxygen was continuously administered through this into the lungs. All anesthetic was discontinued at this stage of the operation.

A large nut was found in the right bronchus and removed. During the operation, the pediatrician had been listening to the heart, and announced that it was in very poor condition, liable to stop at any time. Adrenalin and the usual stimulants were given during the operation.

The above were of no avail, and the patient expired in the operating room, no time being allowed for further search of the bronchi for other foreign bodies which might have been present.

An autopsy was performed, and the following findings were noted:

"The epiglottis and vocal cords are somewhat swollen. The mucous membrane of the trachea is covered with multiple confluent hemorrhages. These are also found in both bronchi and their ramifications. In the right bronchus about midway down, there is a thin piece of nut shell. It measured about 1 by 1 cm. The lungs are very edematous and show scattered areas of atelectasis. There is no consolidation."

"The esophagus is full of ingested food, and the stomach contains more than a pint of food, mostly coarsely chewed nuts."

Case 13.—L. S., a white male, 18 months old, was admitted to the Buffalo City Hospital, October 10, 1933.

Four weeks previously the child had aspirated a bead, and since then had had paroxysms of coughing.

Examination showed a well developed baby of stated age. Apparently the child was not acutely ill. General examination disclosed no abnormalities. The lungs were resonant throughout, the breath sounds were roughened at the bases, there were no rales or rubs. The heart was regular and rapid. There was no bruit and the tones were good. The pediatrician noted that the examination was not entirely satisfactory because the baby cried continually. The rectal temperature was 99 upon admission, the pulse 130.

An x-ray of the chest showed a large bead in the right bronchus, just below the bifurcation.

The night of admission the baby was very restless, and coughed considerably. The temperature was 99.6 (rectal), and the pulse was 120.

The following morning, October 11, the patient was prepared for operation and given 1/300 gr. of atropine. Bronchoscopy was done under ether anesthesia. The anesthetic was given by a competent anesthetist, and though entirely safe, was a little deeper than desired with so small an infant. An attempt to pass a 5 mm. bronchoscope was unsuccessful, owing to a very small glottis and overhanging aryepiglottic folds. A 4 mm. bronchoscope was then passed, but a forcep small enough

to pass through this instrument was not large enough to hold the bead while passing the cords. The bead was twice brought up to the vocal cords, and lost at this point.

The operation was then postponed until a fenestrated forceps capable of passing through a 4 mm. bronchoscope was obtained. The operation took about 8 minutes, and the child left the operating room in good condition.

After returning to bed, the patient came out of the anesthetic normally, but was very restless. It was difficult to keep him in bed, and the mother was allowed to hold him.

At 7:00 that evening the patient started to have respiratory difficulties. Respirations became rapid and labored, and the nails were slighty cyanotic. He was immediately placed in an oxygen tent. The color improved slightly and the pediatrician's examination at this time showed both sides of the chest crowded with expiratory and inspiratory rales. The heart was rapid, regular, toxic, and there was no bruit. It was believed that the child had a passive congestion of the lungs, with early bronchopneumonia, and toxic myocarditis.

The oxygen was continued. 1/250 gr. of atropin was given, and fluid was administered subcutaneously. At 7:45 the patient became more cyanotic and rapidly grew worse. Since there were no signs of laryngeal obstruction, tracheotomy was not considered. At 8:30 respirations ceased. The temperature just before death was 110 (rectal).

The mother confirmed our observations of the small larynx, saying that during the first few months of the baby's life, it had had very noisy respiration.

Case 14.—J. M., a white female, aged 10 months, was admitted to the Buffalo Children's Hospital, June 30, 1936.

On June 24 the child had been given some peanuts by her older brother. Finding the baby cyanotic and choking, the mother turned it upside down, and part of a peanut was coughed out. The choking spell ceased, and the child appeared all right, except for occasional coughing and wheezing. This persisted during the day, and the family physician was called. He kept the baby under observation for six days. The child never appeared very ill during this time, but as the cough persisted, she was brought to the Children's Hospital for an x-ray of the chest.

An x-ray examination showed an area of atelectasis in the lower right lung. There was also interstitial emphysema of the neck. No shifting of the mediastinum, or collapse or emphysema of the lungs was observed. The findings, however, were very suggestive of a foreign body in the lower right bronchus.

The child was admitted to the hospital.

At the time of admission, the temperature was 101.2, the pulse 130, and the respirations 30. A white blood count showed some 18,700 cells, of which there were 56 polymorphonuclears, 44 per cent lymphocytes. Examination revealed a well nourished, well developed child, breathing with difficulty, and coughing considerably. The physical examination was negative except for the chest. Impaired motion over the right lower chest, and dullness over the right lower lobe were revealed. Breath sounds were diminished over the right lower lobe, and the heart was shifted slightly to the right. There were no murmurs.

The patient was bronchoscoped without anesthesia. About one half of a peanut was removed from the lower right bronchus. This bronchus contained consid-

erable muco-purulent secretion, which was aspirated. Further search revealed no more foreign bodies. The operation lasted a very few minutes, and the child left the operating room in good condition. She was immediately placed in an oxygen tent.

The patient's temperature went up that day to 103, and the pulse rose to 140. The respirations stayed around 30, but were somewhat labored. On the second day postoperatively, the temperature stayed at 101 (rectal), the pulse remaining at 140, and the respirations staying around 40 for a minute. The patient took fluids and milk without difficulty, and the color remained good. She had a quiet night.

The third postoperative day there was a sudden change, the temperature suddenly rising until it stood at 107 that afternoon. The pulse and respirations went up accordingly.

An x-ray taken at this time showed an area of atelectasis in the lower right lung, a considerably smaller extent than the first time. There was still bronchopneumonia in the upper right lung.

The child expired that night.

An autopsy was performed, and the following notes were taken from the report:

"A ten months old white baby girl, in a well nourished condition. There is a pale, anemic tinge to the skin. Deep in a secondary bronchus, leading to the posterior and medial part of the right lower lobe, is a small portion of peanut. There is considerable purulent bronchitis of the right lower lobe. There is a distinct collapse with lobar pneumonia of the entire right lower lobe and the upper posterior third of the right upper lobe. A slight hyperemia of the right tracheobronchial lymph nodes is noted. There is a marked compensatory emphysema of the left lung and the right middle lobe. Distinct interstitial emphysema of the latter lobe, and very marked emphysema of the posterior mediastinal tissue is present. There is a plum sized bleb between the esophagus and the azygos vein."

The foreign body was imbedded so deeply into the small bronchus that it was at first overlooked by the pathologist, and only came to light when the lung was compressed by the hands.

CASE 15.—F. W., a white male, aged 10 months, entered the Buffalo City Hospital, April 7, 1936.

Twenty-four hours before admission, the family physician was called to see the baby, who was coughing violently. The mother had not seen the child choke on anything, but he had been coughing severely for several hours, at times becoming blue. When the doctor arrived, the patient seemed much better, but he brought the baby into the Buffalo City Hospital for observation the following day.

Examination of the chest by the pediatrician showed scattered areas of bronchopneumonia. The temperature at the time of admission was 100, the pulse 150, and the respirations 48.

An x-ray of the thorax showed evidence of obstructive emphysema on the right side, with a displacement of the heart to the left. The roentgenologist expressed the opinion that a nonopaque foreign body in the right main bronchus, such as a peanut, should be considered.

The patient's general condition was very poor, the breathing labored, and cyanosis was present to a moderate degree.

Bronchoscopy was done without anesthetic. A 4 mm. bronchoscope was used, and upon entering the trachea, a large foreign body, which later turned out to be a portion of a dried kidney bean, was observed and grasped with a fenestrated blade peanut forceps. Because the foreign body had enlarged since its aspiration, from the moisture and heat, it became stuck in the vocal cords, pulling loose from the grasp of the forceps. The child immediately became very cyanotic, and a quick removal of the foreign body was necessary. The bronchoscope was again passed in search of further foreign bodies. In all, eight large pieces of bean were removed from the trachea and bronchi, three of which stuck at the vocal cords, necessitating quick removal after withdrawal of the bronchoscope. After the last foreign body had been removed, the tracheobronchial tree was carefully examined for smaller fragments, and the retained secretions were aspirated. The operative measures required about 15 minutes and oxygen was continuously administered through the bronchoscope during this time.

Because of the unusual trauma of the vocal cords, resulting from the large foreign bodies, we were on the lookout for laryngeal edema, which began to appear in about 12 hours. This necessitated a tracheotomy, after which the child appeared to improve. During the course of the next two days, the temperature and pulse gradually mounted, the temperature reaching 105.4 (rectal) 48 hours after the operation. An examination of the chest posteriorly showed areas of tubular breathing throughout both sides and patches of consolidation in the left upper and infrascapular regions. The heart tones were only fair.

On the evening of the third day the child became cyanotic, and breathed only at long intervals. There were muscular twitchings of the fingers and toes. The patient was hurried to the operating room.

A bronchoscopy was immediately performed. Both bronchi were full of a thin, watery secretion, which was aspirated at short intervals through the bronchoscope, but seemed, nevertheless, to well up from the smaller bronchi in enormous quantities. Oxygen was passed directly into the lungs through the side channel of the bronchoscope.

The baby appeared much improved after this treatment, but only for a short time, and expired 24 hours later, with a clinical diagnosis of pneumonia.

An autopsy, limited to the chest, was secured. There was no sign of infection around the tracheotomy tube. There was no pneumothorax. The pleura was not remarkable. The lungs were slightly smaller than normal, gray in color, with many reddish purple patches irregularly distributed throughout. These were concentrated on the dorsal aspect of the upper portion of both the right and left lower lobes. They were in part friable and soft; also, noncrepitant, and partially nonyielding. On section, some such areas presented a very granular pneumonic surface, while others were nongranular. The entire bronchial tree was very carefully dissected. There was no foreign body present. The bronchial mucosa was blood stained and hemorrhagically congested, containing a moderate amount of thin, mucoid fluid. The bronchi were all dilated and thin walled. There was no sacculation or fusiform dilatation, but rather, the dilatation was uniform throughout. The remainder of the lung parenchyma was normal in appearance. The lymph nodes of the bifurcation of the bronchi were reddened, but otherwise not remarkable. The pericardium was negative. The myocardium was ruby red in

color, fairly firm, and not friable. There was no pathology of the valve or orifices. The tricuspid ring was 1 cm. larger in circumference than the mitral.

COMMENT

I. DIAGNOSIS

It is quite evident after reviewing the history of these cases that a prompt diagnosis of the presence of a foreign body is imperative, if our mortality percentage is to be kept at a minimum. True, a foreign body may occasionally remain in a bronchus for years. Simpson[2] mentions a case with a foreign body in place for nine years. Vinson[3] reports that a peanut was spontaneously expelled from the bronchus after one year. As a rule, however, such cases come to grief much earlier. Jackson[4] discloses that only about 2 per cent of foreign bodies are coughed up. This is in line with Gill's[5] statement that "expectant treatment is always dangerous."

Of the fifteen cases here presented, a faulty and delayed diagnosis was made in eight. Of the eight cases, a faulty diagnosis in six resulted in such delay that death was inevitable. In the other two cases, both very young infants, the delay contributed to such a condition of weakness that an unfavorable outcome was greatly facilitated.

It is not our purpose to give full discussion of all the factors of diagnosis, especially the many clinical signs, but rather to comment upon and emphasize certain proceedings that must be carried out if patients with foreign bodies are to come to operation in a reasonable length of time. Jackson[1] has well said "Failure to make the diagnosis is due not so often to inability to make it as to failure to consider it as one of the diagnostic possibilities."

It is rather surprising to see the number of cases of faulty diagnosis, entirely resulting from the lack of a good history. Many times the complete story of choking on a peanut, or "swallowing" a bead, is there for the asking. Many of these children come from poor and ignorant families, and the parents do not realize the significance of the history or think to volunteer the information. In a large city hospital many parents are of foreign extraction, and, not understanding the interne who is taking the history, give misinformation. In reporting on a series of twenty-six nut cases, Lyman Richards[6] found that the history was negative in two cases, and in five cases was obtained only by direct questioning. The latter figure is quite significant. Ginzburg,[7] in reporting on pulmonary suppuration due to aspiration of foreign bodies, observes: "The recognition of the cause of suppuration in these cases depends primarily on the history and

the slow, insidious development of the process frequently interpreted as a recurring pneumonia." In some cases, however, a definite history of aspiration of a foreign body is never obtained. Clerf[8] has fittingly remarked, "It is unwise to conclude that the symptoms are not due to a foreign body, because the patient has no recollection of having swallowed a foreign object." Lawrence Jackson[9] has said, "Foreign body must always be thought of in the diagnosis of chronic pulmonary disease, whether or not there is a suspicious history."

Cases 1, 2 and 10 in our series gave no history of foreign body aspiration. Case 2 did not give a history at the hospital because for many years, as he said, he had told doctors about swallowing a bone, and since the x-rays were negative and the physicians failed to attend to that part of his history, he had ceased mentioning it. The asking, in this case, would have established the diagnosis and probably saved the patient's life.

Second in importance to the securing of a good history, and ranking in significance with the physical examination, is the careful x-ray study. The word "study" is used advisedly, for the roentgenologic diagnosis of nonopaque foreign bodies in either the esophagus or the bronchi is the result of careful study of the several small, sometimes minute, deviations from normal which arise from the secondary pathologic changes induced by the foreign body. The percentage of correct diagnosis in these cases will necessarily be much higher in large hospital centers, where these fine points of diagnosis will be observed more frequently. Moore, Wilson and Arbuckle,[10] Reineke,[11] Iglauer,[12] Flynn,[13] Jesberg,[14] Scott and Moore,[15] and Clerf,[16] along with many others too numerous to mention, have all insisted upon such study. We fully agree with Pendergrass,[17] who states that "No patient should be submitted to bronchoscopy without roentgenologic examination." The only exception to this rule would be in cases of great emergency, where immediate operation upon a patient with an obvious foreign body is imperative.

The roentgenologic technic for the examination of both the esophagus and the tracheobronchial tree for foreign bodies has been thoroughly worked out in the clinics, notably that of Pancoast.[18] It should be noted, however, that obstructions in the esophagus do not always present the same picture. Partial obstruction due to nonopaque foreign bodies lying in the upper end of the esophagus is sometimes easily missed. Partial obstructions, low down, may at times give a picture closely resembling that which is caused by an intrinsic tumor, or, if properly placed, may be confused with early cardiospasm. Case 6 of our series demonstrates such a case. A very excel-

lent roentgenologist gave as his opinion that the obstruction was not caused by a forign body because of the very uniform narrowing of the lumen. The barium, therefore, seemed to be passing through a very small opening, and the deformity had very smooth outlines.

Sometimes foreign body removal has been considerably delayed because the offender has been buried in a mass of closely adherent barium paste. Bones are often so obscured that careful study of their position in the lumen and location of their dangerous points can only be accomplished after a long and tedious cleaning away of the diagnostic meal. It seems preferable to have the patient swallow a large, barium-filled capsule which will stop at the point of obstruction. The ingestion of lipiodol is more desirable still; enough of it will adhere to the foreign body to outline its location and still not hinder the operation that may shortly follow.

The conditions of "air-trapping" caused by foreign bodies in the bronchi are now well understood. Both the unilateral and bilateral emphysema cases have been discussed thoroughly in modern literature, and all roentgenologists are acquainted with their significance. However, in cases where the secondary changes due to nonopaque foreign bodies are early and exist in only slight degree, a technic of the highest quality must be used. A small lag in one diaphragm, a very slight shift of the mediastinum, seen in an anterior-posterior view taken without any rotation, a slight narrowing of the intercostal spaces, a slight difference in the degree of aeration of one lobe compared to the other—these are all most important in the diagnosis of the obscure cases.

When even the most careful and expert roentgenologic examination fails to show evidence of a foreign body, it should not be accepted as final proof, if the history or clinical findings in the case point to the possibility of a foreign body. Clerf,[16] Ginzburg,[7] Scott and Moore[15] and others have expressed this view.

In our series three cases had had x-ray studies, in which no foreign body was found. In Case 1, it was a small stone or piece of shell, and in Cases 2 and 3 there were rather large bones. There was ample justification for bronchoscopic examination, even without the x-ray finding of a foreign body. In fact, as previously observed, Case 2 stopped mentioning the fact that he had "swallowed" a bone because the "doctors could not find it in the x-ray." In Case 1 there was very little indication for suspecting a foreign body, and a positive x-ray finding before her final attack of severe hemoptysis would have indicated bronchoscopy, probably saving the patient's life.

The third method of diagnosis, the physical examination, is generally the most difficult. All physical signs are dependent upon the type, location and degree of obstruction, and therefore vary markedly from one to another. They also vary and change greatly during the length of time that the foreign body remains in place. Examination is also modified by the material that has been aspirated. It is well to remember that signs may occur in the opposite lung, in which no foreign body may be present. Hara[19] has shown in his work on rabbits that pathologic changes often occur in the lung opposite to the one containing the foreign body.

Varying degrees of dysphagia and pain are the cardinal symptoms of esophageal obstruction. Bronchial obstruction gives more numerous findings, the most characteristic being limited expansion, decreased vocal fremitus, impaired percussion note and diminished intensity of the breath sounds distal to the foreign body. The symptoms of cough, asthmatoid wheeze, tracheal slap and occasionally pain are generally present, although not all in one patient. As in all other types of pathology, the clinician who has had experience in examining many of these cases will become more expert in diagnosing them, even without a leading history, than another equally proficient but inexperienced diagnostician. The technical bronchoscopist will seldom rely on his own stethoscope, but can aid best in consultation with the roentgenologist and internist by applying his own peculiar judgment gained over a long experience with these cases.

II. MANIPULATION

When a child aspirates a foreign body in the presence of his parents or other folks, they generally make a hasty attempt to extract the offender with their fingers. These attempts are generally ineffectual and have at times added difficulties to the removal of the foreign body. In Case 8 of our series the mother, in an attempt to recover the many pointed religious medal from the child's throat, succeeded only in pushing it down and impacting it so tightly that removal from above was impossible.

Years ago a favorite method of physicians in cases of foreign bodies of the esophagus was to push the offender down with a stiff stomach tube or even with a stiff bougie. Jackson,[1] Clerf,[5] Orton,[20] Patterson[21] and others have repeatedly called attention to the danger of these procedures until now it is rarely done by the examining physician who is first called. None the less, an occasional case presents itself, as Case 7 of the series, where an attempt had been made to push an open safety pin into the stomach. The result was that

there was a perforation of the esophagus by the time the patient reached the hospital. Several patients who recovered and are therefore not reported here had had previous manipulation before coming under our observation, and this proved a great factor in increasing the danger of the operation. One cannot urge too strongly the elimination of preliminary meddlesome treatment in such cases. In addition to this, the old method advocated by Lebensohn,[22] of giving apomorphin to expel food impacted in the esophagus, would seem very dangerous. Rupture of the esophagus during violent vomiting is by no means rare, and I believe that general adoption of the above procedure would make it a frequent occurrence.

III. IMPROPER PREPARATION OF A PATIENT

The proper preparation of a patient for removal of foreign bodies is as important as in any other major operation. The stomach should be empty. Emesis during operation not only obscures the field but adds danger of the aspiration of other foreign bodies. The proper dose of atropin should be given time to produce a field as free of mucus as possible. If very recent unsuccessful endoscopy has been done it is well, if possible, to allow the patient to rest a few days before attempting operation, especially in the case of an infant where the foreign body is not causing immediate danger.

In the case of a suspected or known perforation of the esophagus it is well to defer removal of the offender, putting the esophagus at rest and supplying external drainage, if necessary. Early operation of these cases is liable to break down the forming barriers to the infection and thus change a controlled infection into a fulminating one. Naturally, every case is a law unto itself, and no hard and fast rules may be laid down. Occasionally, as in Case 11 of our series, the emergency is so great that immediate operation is imperative, and no time is permitted for proper preparations. On the other hand, just because the patient has been rushed to the hospital, and the parents or friends have been told by others that an operation should be done at once, is no reason for the endoscopist hurrying into operating without a careful planning of all preparation details for the patient and the operating room. All instruments that may be needed should be available. If the bronchoscopist is used to working with a specially trained personnel, as he should be, time should be taken for the assembling of that personnel.

IV. EXTREME YOUTH OF THE PATIENT

As in most illnesses, an infant or small child can stand much less than an older patient. Infancy is a very important and un-

controllable factor in the mortality of foreign body cases. The operator must take less time for the operation, be more gentle in the handling of his instruments, use less or no anesthetic, be handicapped by using smaller tubes, and then, after providing for all these prerequisites, stand more chance of fatality, due entirely to the patient's age.

In our series, ten cases were operated upon. Seven of these ten were 18 months or younger, the average age being 13.5 months and the mean age 13.3 months. The three other patients were 9, 10 and 68 years of age respectively. The last two were first seen when they already had fatal complications, and the first case was in a most critical condition when first seen. Of the seven cases under 18 months of age, only one had a fatal complication when I first saw him. The other six were not hopeless when they came under my examination and should not have died if everything had gone well. Having seen many cases in much more critical condition than any of these, I believe that five of them would have been saved, but for the very slight resistance occasioned by their extreme youth. True, all of the above mentioned foreign bodies were technically more difficult to handle than the average, but age was the deciding factor.

Since there is no way to control the patient's age, the operator must bend every effort to assure an organization at peak efficiency when very small children are dependent upon him for delicate surgery.

V. ANESTHETIC

The question of anesthetic in a foreign body case has long been debated. Jackson[1] fittingly observes, "The choice of an anesthetic for each operation must, however, be a matter for individual decision and will depend upon the personal equations, the degree of skill of the operator and his ability to quiet the apprehensions of the patient. In other words, the operator must decide what is best for his particular patient under the conditions then existing." Wood[23] believes that bronchoscoping children without anesthetic is ideal, but was able to do much better himself when ether was used. Miller[24] gives no anesthetic to children under seven years of age. For patients from seven to fourteen years of age he uses a general anesthetic. Boot[25] does not believe in an anesthetic for small children. Vinson[26] has employed ether in many cases, but concludes that it should be given to children in esophageal cases only, and to adults with large foreign bodies in the esophagus. For tracheobronchial foreign bodies in small children he gives no anesthetic. Richards[6] feels that no anesthetic

is better in children, but advises the use of avertin by less skillful operators. Richards had no trouble with avertin, and the only death in a series of 26 nut cases was a child operated upon with no anesthetic.

In accordance with the majority, I feel that tracheobronchial foreign bodies in small children should be removed without anesthetic. In children, six years old and over, however, especially those with nonvegetal foreign bodies and no marked lung improvement, a light gas-oxygen-ether anesthetic, given by a specially trained anesthetist, is safer than being handicapped in the operating room by the violent struggles of the child. The operation can, as a rule, be performed more quickly, with less shock and trauma. It also permits the anesthetist to keep a constant stream of oxygen insufflating into the lungs through the side channel of the bronchoscope. This has proved a decided benefit in some of our cases. I am firmly convinced that such an anesthetic, skillfully controlled by a trained man, is a decided advantage to the operator and the patient. Avertin used in doses considered safe for children has proved of no advantage. An additional anesthetic is always necessary. It is desirable to have the patient awake and coughing as soon as the operation is completed, and since avertin does not permit this we have discontinued its use.

Only in one of our cases, Case 13, do we believe that the anesthetic may have been partially responsible for the unfortunate outcome, and then only because we are at a loss to explain the child's sudden death. As a matter of fact, we have seen the same pulmonary edema occur quickly in children operated upon without anesthetic, viz, Case 15. Wood[23] has pointed out that pneumonia, often a complication when a general anesthetic is used, cannot always justly be traced to the anesthetic. On the other hand, we believe that a general anesthetic is Case 8 might have relaxed the cricopharyngeus enough to permit removal of the foreign body.

A local anesthetic is used for all adults, with the exception of those who are difficult to handle and have large, dangerously pointed foreign bodies in the esophagus. In these cases the relaxation desired is achieved by ether anesthesia. In all other cases it has been our custom to use cocain in sufficient quantity to attain a good anesthesia. The usual pantopan and atropin clinic hypodermic is given. Of late, decidedly good results have been obtained with the use of 2 per cent pontocain, and we have gradually discontinued the use of cocain, although none of our cases have ever showed even the slightest symptoms of cocain poisoning.

VI. SPECIALLY TRAINED PERSONNEL AND PROPER INSTRUMENTS.

Competent, trained personnel and proper instruments are exceedingly important factors in the prevention of mortality in foreign body cases of the esophagus and tracheobronchial tree. Undoubtedly many an operator has been just on the point of capturing a small piece of peanut buried deep in bubbling pus, when the head suddenly moved, the wrong instrument was handed to him or the mouth gag slipped, allowing the patient's teeth to move the bronchoscope. These and a number of other mishaps may occur at a critical moment, delaying the operation and permitting the small foreign body to escape into a small bronchus, never to be seen again.

Often there is no time so opportune for removing a foreign body as the first time it is sighted. All the mishaps mentioned above are entirely chargeable to the operating room personnel. To eliminate these during operation it is well to have one or more assistants who are specially trained to hold the head. Dependence upon hospital internes is nothing short of optimism. Proper head holding is not learned in the course of an operation, and internes are changed too quickly in service to ever permit their proper training. And, after all, they are generally not interested in such duties.

It is also very important to have one nurse in the clinic who is directly in charge of the instruments and capable of handling them at the time of operation. Those operators who have large city bronchoscopic clinics will have the organization called for. In smaller cities, however, specially trained operating room personnel may be lacking and can never be entirely offset by the skill of the operator.

The importance of a sufficiently large and varied collection of instruments cannot be too strongly emphasized. In most cases the patient's condition will permit the time necessary to send to the manufacturers for the required instrument. However, this is never desirable and in critical cases may decidedly influence an unfavorable result. This type of hazard is well illustrated in Case 13 of our series. A bead forceps large enough to grasp the bead and still pass through a 4 mm. scope would very probably have saved the child's life. Ellen Patterson[27] has had a similar experience.

The great variety of objects that serve as foreign bodies and the diverse conditions attendant in such circumstances demand an extensive instrumentarium. This requires such a considerable outlay that the list of instruments in the smaller clinics may be inadequate to every occasion. In this instance, the preferences of the various operators are again emphasized. Meyers,[28] Yankauer,[29] Chamberlin[30] and

Cunningham[31] occasionally use hooks for beads, but Patterson[27] thinks that these may be dangerous Jesberg[32] devised a method for the removal of beads with a stylet and rubber band. Whatever the surgeon's choice, it must be at hand for immediate use if regrets are not to follow.

The instruments must be of the best quality, and even these will deteriorate without proper care, possibly breaking, to give a foreign body where none was before. Jackson[33] has reported a sizeable list of the results of such accidents.

An operator in a large city may at times be called upon to operate in a hospital with insufficient equipment. In this case it is undoubtedly safer for him to take his own assistant at least. Dependence upon ordinary hospital routine, even in anticipated easy cases, will sooner or later result in regrets. Although none of our operated fatalities in the above series can be even remotely attributed to anyone but myself there have been other cases, fortunately not ending in fatality, which amply justify the above observations.

VII. THE PROPER JUDGMENT IN THE USE OF TRACHEOTOMY

The proper use of tracheotomy is an important factor of safety in foreign body cases. Naturally its use in any long series of cases will depend greatly upon the judgment of the operator. In early years this operation was done to facilitate the removal of the foreign body. There are still some men who advocate inferior bronchoscopy as a method of choice, e. g., S. Belinoff,[34] who believes that inferior bronchoscopy should always be done in children under the age of six. However, Jackson[1] says, "Tracheotomy is no longer indicated either for the removal of the intruder or for the insertion of the bronchoscope." With the last statement, I think that the majority of the authorities agree. There is possibly one type of case in which inferior bronchoscopy might be a distinct advantage over a peroral operation. When beans are aspirated they quickly absorb moisture and increase in size. A large kidney bean may go through the glottis easily enough when aspirated, but after some time in the warm, moist air of the tracheobronchial tree it may fail to come back through the glottis so easily. We very nearly lost such a case on one occasion. The foreign body could not be grasped tightly enough, short of crushing it, to bring it back through the glottis, and we resorted to inferior bronchoscopy for its removal. Since the patient was in a critical condition when first seen, the delay almost resulted in a fatality.

The important use of tracheotomy in foreign body operations comes in the postoperative care of the patient or in the pre-operative

stage if laryngeal obstruction is present. Vinson[26] has indicated that it is very difficult to tell at the time of bronchoscopy whether laryngeal edema will develop, and if so to what extent. A small child may show no laryngeal effects after a long and difficult operation, while, conversely, a large child may develop laryngeal obstruction a few hours after a short operation. There is no doubt, nevertheless, that careful handling of small tubes through the glottis will cut down the incidence of laryngeal edema to a minimum.

Not all cases of laryngeal edema will necessitate tracheotomy. Vinson[26] reported twenty-nine cases of tracheotomy in ninety-seven children under the age of five who had tracheobronchial foreign bodies removed. He stated that undoubtedly some of the cases would have recovered without the operation, but that it was far better to open the trachea as soon as breathing had become definitely obstructed than it was to wait until such time as the tracheotomy would have to be done as an emergency measure. There can be no criticism of this viewpoint.

Jackson[1] also advises tracheotomy when a child is drowning in its own secretions, thereby permitting the attendant to aspirate frequently through the tube. This was done in Case 11. The child had a trained, special nurse throughout its illness to give the proper post-tracheotomy care—all to no avail. Had the same procedure been adopted in Case 15, recovery might have occurred, but that is exceedingly doubtful. We do believe, however, that Case 12 might have been saved if tracheotomy had preceded bronchoscopy. The foreign bodies could have been removed by inferior bronchoscopy without the delay and difficulty occasioned by the child's repeated vomiting.

VIII. PERFORATION OF THE ESOPHAGUS

Mortality resulting from foreign bodies of the esophagus is practically always due to a perforation of the thin esophageal wall which in turn creates an infection of the peri-esophageal spaces. Boot,[35] King,[36] Mullin,[37] Tucker[38] and others have reported fully on such cases. The type of perforation and its particular location in the esophagus decidedly influence the outcome of the case. Furstenberg[39] and Lambert and Berry[40] have demonstrated that for a thorough understanding of the chronologic chain of events occurring after perforation, a satisfactory knowledge of esophageal anatomy and its relations is imperative.

For all practical purposes we may consider the four fascial planes in the neck. Each of these planes gives off numerous side sheaths

which support various organs and muscles, but do not form the boundaries of the broad highways to the mediastinum. It is these highways that are utilized by acute infections of the peri-esophageal spaces.

The Superficial Layer.—This layer is rather thin and completely surrounds the neck, being attached posteriorly to the ligamentum nuchæ, and splitting at either side to invest the trapezius and then the sternocleidomastoid muscles. The two halves meet in front at the midline. This layer is attached above to the occipital protuberance and the mastoid bone. It merges with the parotid gland capsule and extends along the lower border of the mandible, afterward coursing downward to about one inch above the sternum, where it splits into two layers, one attached to the posterior, the other to the anterior surface of the sternum.

The Pretracheal Layer.—This layer lies in front of the trachea, over which it sends a small process anteriorly to blend with the superficial layer. Laterally it passes beneath the sternohyoid, omohyoid and sternothyroid muscles and then splits to invest the thyroid gland. Laterally to this it blends with the carotid sheath and the layer of superficial fascia that passes posteriorly to the sterno-cleido-mastoid muscle. Above, the pretracheal fascia is attached to the hyoid bone and cricoid cartilage. Below, it passes to the aorta and the pericardium. At the entrance to the thoracic cavity it sends a process anteriorly to join with the sternum, thereby shutting off the mediastinum from a descending infection using this pathway.

The Buccopharyngeal Fascia.—This is a thin layer of connective tissue which invests the superior constrictor muscle, thus forming the anterior wall of the retropharyngeal space. Laterally it merges with the pretracheal fascia. Superiorly it runs up to the buccinator muscle; below, it extends down behind the esophagus to the posterior mediastinum.

The Vertebral Fascia.—This layer covers the bodies of the vertebrae and the vertebral muscles. It extends laterally to form the carotid sheath, and then laterally to cover the subclavian artery, scalene muscles and branchial plexus. Above, it attaches to the base of the skull, and continues down into the posterior mediastinum. It is evident that the deep fascial layers form three highways, as it were, leading from the upper cervical region down to the mediastinum.

1. The previsceral space.—This pathway lies between the superficial and the pretracheal fasciæ.

2. The visceral space.—This space lies between the pretracheal and the buccopharyngeal layers, and holds the trachea, the thyroid gland and the esophagus.

3. The retrovisceral space.—This pathway lies anteriorly to the prevertebral fascia and is bounded anteriorly by the buccopharyngeal fascia.

At this point another main pathway for infection must be considered: The carotid sheath, which is a firm, dense, fibrous, fascial envelope, investing the great vessels of the neck. The upper portion is the pharyngomaxillary fossa, the importance of which has been sharply stressed by Mosher.[11] This fossa is a small funnel shaped space, high in the neck, bounded above by the base of the skull, laterally by the ramus of the mandible and the parotid gland, internally by the superior constrictor muscle and posteriorly by the upper cervical vertebra. A penetrative wound of the lateral hyopharynx may carry infection directly to this space, and the resulting infection can easily migrate downward into the carotid sheath, where infectious thrombosis of the jugular vein may easily result, or a further extension of infection into the posterior mediastinum.

A brief consideration of the mediastinum is in order, for it is the infection in this area that leads to the immediate cause of death. This space may be divided into three compartments:

1. The anterior mediastinum, which lies between the sternum and the pericardium.

2. The middle mediastinum, which contains the heart and great vessels.

3. The posterior mediastinum, which lies between the vertebral column and the pericardium.

It has been proven, both by laboratory methods and by clinical experience, that the visceral and retrovisceral spaces of the neck lead directly to the posterior mediastinum. Furstenberg[39] considers the latter path the most important channel leading to the mediastinum, and has shown by serial section that it exists as a definite space throughout its course to the mediastinum.

The visceral compartment also leads directly to the mediastinum. Furstenberg[39] reports a case where lipiodol was injected into this space during an attempt to instil it into the trachea. The course of the oil was followed roentgenologically downward by the visceral pathway into the posterior mediastinum.

The anterior mediastinum seldom is infected from above. The previsceral space is blocked off from it by the above mentioned fibrous sheath, extending from the pretracheal fascia to the posterior surface of the sternum.

Two main methods of approach have been worked out for drainage of a mediastinal abscess, and there seems to be no universal agreement upon the preferable method. First, there is dorsal mediastinotomy, in which the mediastinum is opened posteriorly by resection of a rib or removal of one of the transverse processes with a portion of rib attached. Secondly, there is cervical mediastinotomy, in which the posterior apartment is reached from the lower cervical region. Most thoracic surgeons prefer this operation if the origin of the infection is above the fourth thoracic vertebra. When pus enters the mediastinum from the neck, cervical mediastinotomy seems the preferable course.

There comes a time to every endoscopist when he must decide what to do in a case of known perforation of the esophagus. He may either know of the perforation by seeing it through the esophagoscope or by the position of a large foreign body in the esophagus, as revealed through the instrument or by x-ray.

Heatly and Pearse[42] have described four types of perforations:

1. "A perforation may occur slowly by erosion. Sufficient time is given before actual perforation, for inflammatory reaction around the lesion to wall off the pathways to the mediastinum; a localized periesophagitis results. This may rupture back into the esophagus, and Nature may thus do the work that the surgeon might otherwise attempt. These cases do not as a rule present a stormy course. The patient may have localized pain in the neck, some dysphagia, a rise in temperature, and occasionally, some subcutaneous emphysema. As a rule, spontaneous healing may take place. Occasionally, the endoscopist will open into the abscess through the esophagoscope, thus bringing about a cure."

2. "A perforation may be minute, as from a pin. Infection is carried into the periesophageal spaces and becomes walled off. After a few days of very little activity, it suddenly starts to extend, and death results from mediastinitis or septic thrombosis. These cases are very insidious in the early stages, and quite often give no warning of the fatal outcome."

3. "The third type is a perforation of the posterior wall. This is very serious as a rule, and deeply placed, infection going quickly to the mediastinum."

4. "The fourth type is the large, tearing perforation that is sometimes the result of the false passage of the esophagoscope in the attempt to remove the large impacted foreign body. In these cases, the patient generally suffers from great pain and shock. Pulmonary collapse may result, as well as emphysema or hemorrhage. As a rule, the patient has no chance for recovery, and expires in the course of a few days."

When such a complication as perforation of the esophagus arises the endoscopist has no set or standard procedure to follow. In the case of perforations of the first three types he may elect to attempt drainage of the abscess through the esophagoscope. Berry,[43] Carmody,[44] Tucker and Wolferth,[45] Meyersburg,[46] Watson-Williams[47] and Imperatori[48] have described such cases, some with success. It would seem that opinion among those with a wide experience, such as Lerche,[49] McGinnis[50] and Heatly,[51] favors the external operation. At present general opinion is most divided on the question of whether to operate as soon as it is known that a perforation exists or to wait until very definite clinical signs and symptoms are present. McGibbon and Mather,[52] after thorough consideration of their own experience, conclude that

1. "Endoscopic methods are justifiable in the attempted drainage of a peri-esophageal abscess, which is due to an eroded perforation."

2. "Known perforations with peri-esophageal emphysema but without general septic symptoms require no active treatment."

3. "Cases with known perforations and signs of general sepsis must be treated surgically."

Iglauer and Ransohoff[53] say, "When the diagnosis of perforation has been established, immediate operation is indicated." Orton[54] believes in waiting until the impacted area has been walled off and the esophagus put at rest by gastrostomy before doing mediastinotomy. Lederer and Fishman[55] suggest that prophylactic mediastinotomy may offer better prognosis than we are now able to give. Sixteen years ago Schlemmer[56] advised early operation as soon as emphysema appeared. Myerson,[57] on the other hand, concludes that external operation may be deferred even in the presence of severe systemic reaction, citing a case to justify his new point. It would seem to me that Heatly and Pearse[42] have given the best indications for the external operation, and we quote them as most nearly meeting the results of our observations.

1. "In cases presenting definite localized abscess of the neck."

2. "In all cases where gross perforation is known to have occurred as the result of faulty instrumentation, prompt external drainage is imperative."

3. "In cases presenting x-ray evidence of a foreign body outside of the lumen in the periesophageal tissues."

4. "In cases where perforation of the posterior esophageal wall can be established by endoscopic and x-ray study."

5. "Emphysema, demonstrable clinically or by x-ray, in association with rising fever and leucocytosis."

6. "In any case where, in the presence of a known or suspected perforation, a reasonable doubt arises as to the safety of the patient."

True, one cannot always work by rule. Extraordinary cases need extraordinary measures. Moersch and Kirklin[58] report the successful removal by endoscopic methods with fluoroscopic guidance of a foreign body lying entirely in the mediastinum. Ball[59] reports a case of posterior mediastinitis passing upward and breaking into the pharynx. These cases probably help to prove the rules by their exceptions.

Case 8, the child who swallowed the religious medal, would probably have been saved if an external operation had been performed and the foreign body removed by esophagotomy, after it was found impossible to disengage the impacted medal from above. Case 5 had a fulminating mediastinitis when first seen, so that external operation would have been of no avail. Case 10 might possibly have had a chance with mediastinotomy, although the thoracic esophagus was perforated. I believe that it would have been very doubtful, however. Case 15, with the purulent pericarditis, would have failed to benefit from this procedure.

An interesting diagnostic and, in some cases, prognostic point, is the occurrence of subcutaneous emphysema. This phenomenon is not uncommon, either in case of foreign body of the bronchus or the esophagus. Clerf[16] has called attention to the fact that the escape of air into the subcutaneous tissues of the neck and thorax is produced in the same manner in bronchial foreign body as in pneumonia, where it is often present. The violent coughing in either case may rupture a vesicle near the periphery of the lung and permit the escape of air. This is well illustrated in the autopsy performed on Case 14, where a large ruptured bleb was found on the posterior surface of the lung near the azygos vein.

Subcutaneous emphysema is frequently caused by the increased intrapulmonary pressure due to foreign bodies acting as check valves, especially when heavy coughing occurs. Vinson and Moersch[60] have reported a case where subcutaneous emphysema was possibly the result of perforation of the bronchus by a sharp, pointed foreign body. This may happen at times, but in our experience is not as common as the first described method. Prognosis in a case of subcutaneous emphysema produced by bronchial foreign body is not necessarily more grave than in a similar case without this complication.

Subcutaneous emphysema occurring in cases of foreign bodies of the esophagus is practically pathognomonic of perforation. Waldapfel[61] states that emphysema may occur not only as the result of a perforation of the esophageal wall but following injury which merely involves the mucous membrane. In the physical examination of any case of suspected foreign body, especially in the presence of dysphagia,

careful search should be made for the possible presence of subcutaneous emphysema around the cervical and upper tracheal regions.

IX. PROPER POSTOPERATIVE CARE

Proper postoperative care of the foreign body patient, especially the very young one, is a most important consideration. It avails nothing to skillfully and sometimes dramatically remove a refractory foreign body if the patient dies a few days later, possibly through avoidable complications.

Laryngeal edema and indications for tracheotomy have been discussed elsewhere. In addition to the usual good nursing care, indispensable to any patient, there are a few conditions peculiar to these cases that need special consideration.

Oxygen as a prophylactic and therapeutic agent has in recent years come into prominence. It is only natural that it should have been tried in the postoperative care of foreign body cases. Vinson[26] reports that after operation he now gives oxygen to all children under five years of age. In 57 of his patients under five years of age who were not given oxygen, 20 cases developed laryngeal edema and required tracheotomy. Of 40 such cases that were given oxygen postoperatively, only nine required tracheotomy.

Evans[62] advises oxygen during operation and immediately after bronchoscpy. The first is accomplished by directing the gas into the lungs through the side channel of the bronchoscope. After operation the child is placed in an efficient oxygen tent, where the oxygen percentage is kept at from 60 to 70 per cent. This generally suffices, but if not, 100 per cent oxygen is given by mouth. Evans[62] states that in an experience of nearly 1,000 cases of anoxemia due to asthma, pneumonia, cardiac failure, etc., he has seen no ill effects when 100 per cent oxygen has been administered almost continuously for two or three weeks. In most instances he has seen it have a very decided effect in clearing up the pulmonary edema. It has been with the close coöperation of Dr. Evans that a routine and efficient oxygen therapy has been administered to all our bronchoscopic cases under the age of five or six years.

Cases 11, 13, 14 and 15 of our series were given oxygen in an efficient manner, both during and after the operation, without noticeable effect. Oxygen apparently did play an important part, however, in many cases that recovered from a very critical condition.

It is dangerous to use morphin after operation to suppress the cough reflex. Case 11 of our series effectively illustrates the result of

such therapy. The bronchi are very hyper-secretory in vegetal foreign body cases and must be freed from all thick secretion that would possibly obstruct the aeration of the lung parenchyma. Nature provided the cough reflex to do just this, and as a rule, adequately cares for this responsibility. If her efforts are thwarted by drugging, atelectasis and pneumonia soon follow. Tucker[15] reports a case of obstructive atelectasis due to the inhibition of cough by morphin. Jackson[1] has repeatedly warned of this procedure. It is not enough to abstain from ordering sedatives in these cases, but an order should always be written that postoperative tracheobronchial foreign body cases should not have such sedatives.

It has never been our custom to do tracheotomy except for laryngeal obstruction. The example of Case 15, however, would warrant consideration of tracheotomy in pulmonary edema, in order to effect frequent aspiration of the profuse secretion that tends to drown the lungs. In case oxygen does not prevent the occurrence of such edema, it would at least be worthy of trial.

In conclusion, may I say that the above nine factors are not the only ones to be considered. Some other operator may discover even more important factors in a series of fatal cases. I do believe, however, that the above mentioned might be taken into consideration by anyone engaged in these most delicate surgical operations.

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XXXVII

MICROSCOPIC OBSERVATIONS OF THE DEVELOPING PETROUS APEX*

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AND

DOROTHY WOLFF, PH.D.

ST. LOUIS

The gross development of the petrous apex was described last year by Profant[9] (May, 1936). In 1933 Glick[5] presented microscopic studies of the apex of the growing child and adult. The anatomy, pathology and clinical importance of this region have been discussed by Eagleton,[2] Kopetzky and Almour[8], Vail,[11] Wilson,[12] Friesner, Druss et al.,[4] Guild[6] and Jones.[7] It is the purpose of this communication to present clearly the microscopic anatomy of the developing apical region as it appears in various stages from the ten weeks fetus to the seven-year-old child. A table of measurements indicating the rate of growth from infant to adult is also included. According to Profant, "there is never a true petrous apex because the region is shaped like that of a truncated pyramid." "Petrous tip" is the term preferred by Profant. The petrous apex or tip, as presented in this paper, may be defined as that portion of the temporal bone anteromedial to the cochlea.

The material used for this investigation includes microscopic serial sections of fetuses of 10 weeks, 3 months, 4½-5 months, 6 months (premature), 7 months and term; infants of 2 days, 10 days, 2 months, 3 months, 5 months, 7 months; children of 4, 5, 6, 7, 8, 10½ and 15 years—and 5 adults. Gross dissection was done of a 5-months fetus (Fig. 3). Routine serial sections of the temporal bone were not included in this study because the complete apex does not routinely appear in the material. With one exception (No. 5563), only that material was used in which the prosector cut sufficiently far anteromedially so that the contour of the petrous apex could be seen in the sections. All material used was cut in the horizontal plane with the exception of No. 5017. This specimen was cut vertically in

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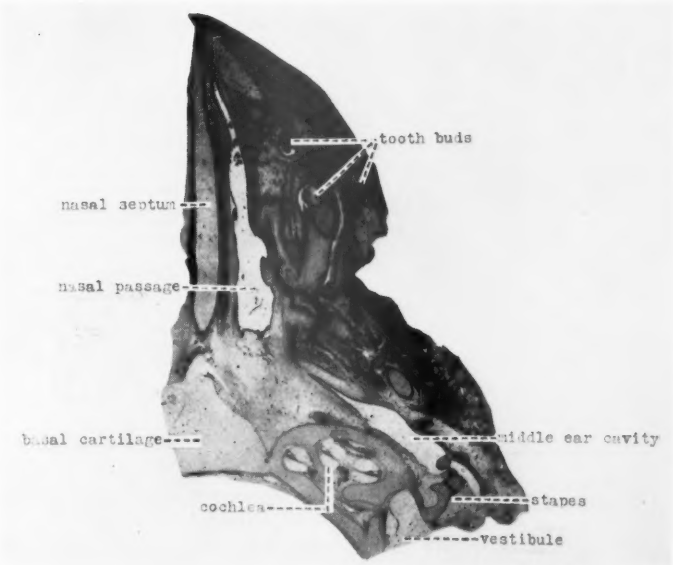

Fig. 1. No. XV M 256, ten weeks' fetus. Section of head showing cochlea in juxtaposition with basal cartilage of the skull. No apex exists at this early age.

the plane of the long axis of the temporal bone. Measurements were made on sections cut as nearly as possible in the plane of the mid-modiolar axis of the cochlea and were taken from the contour of the basal turn forward to the most apical limit of bone. We were in most cases unable to include both ears in the study since frequently the mate happened to be cut vertically or was not cut far enough anteriorly. The reader should remember that nearly all infant laboratory specimens are diseased. This fact should be considered in drawing conclusions on an anatomic basis.

Profant stated, "until the seventh fetal month there is no petrous tip, hence the cochlea is at the apex." The microscopic section of the ten weeks fetus (Fig. 1) shows clearly the absence of any structure anterior to the cochlea. The basal cartilage of the skull is continuous with that surrounding the basal turn of the cochlea. The reader's attention is called to the studies of the chondrocranium as illustrated in the anatomy texts of Morris and Gray. In these works

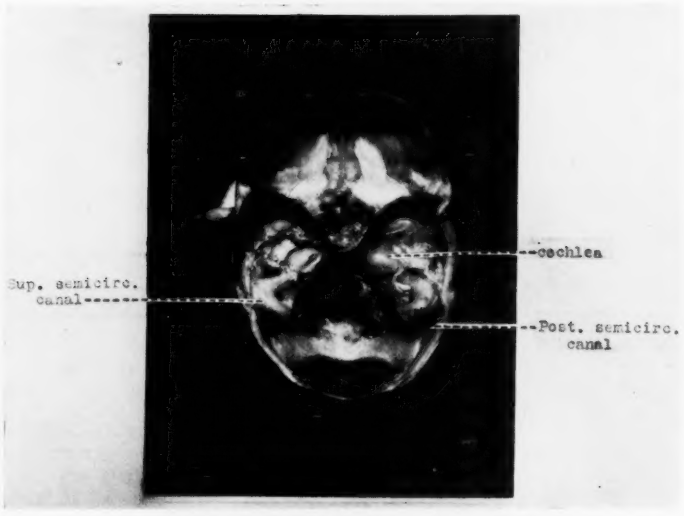

Fig. 2. Gross specimen, five months' fetus. The otic capsule comprises the whole of the petrous bone. No apex has developed.

the reconstructions of the chondrocranium by Lewis, Macklin and Hertwig are well shown. All the fetal stages represented reveal the juxtaposition of the cochlea and the basal skull cartilages.

The microscopic section of a 3 months fetus (Fig. 3) indicates practically the same anatomic condition. At this stage, however, some evidence of demarcation between the otic capsule and basal cartilage has appeared.

In the 4½ months fetal specimen (borrowed from Dr. L. K. Guggenheim's collection) the demarcation between the otic capsule and basal cartilage, as seen under the microscope, is definite. Centers of ossification of the otic capsule are obvious. Bast has shown that at this age ossification centers one and eight, as designated by him and which enclose the basal turn of the cochlea, are approaching each other. At 19 weeks these centers of ossification are united.

Fig. 2, as cited above, illustrates conditions found in a 5 months fetus dissected grossly. Here again the cochlea appears in an antero-medial position with no apex existing. The superior semicircular

Fig. 3. No. 21, three months' fetus. Note close juxtaposition of cochlea with basal cartilage also at this age. No petrous apex has developed.

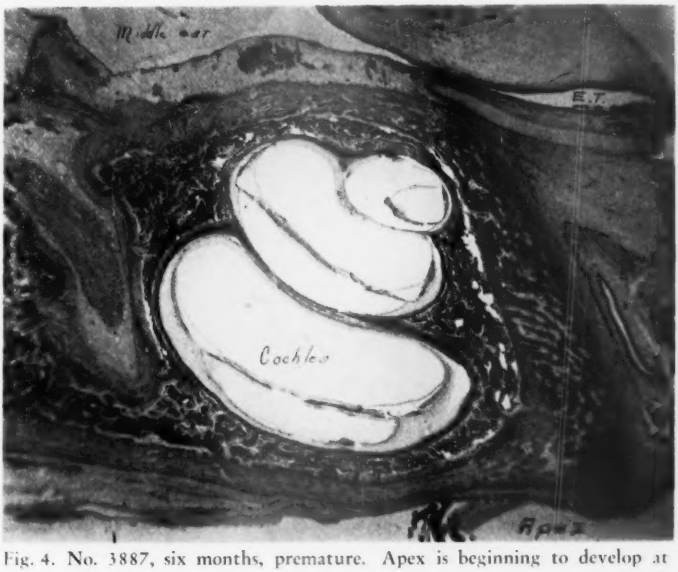

Fig. 4. No. 3887, six months, premature. Apex is beginning to develop at this age.

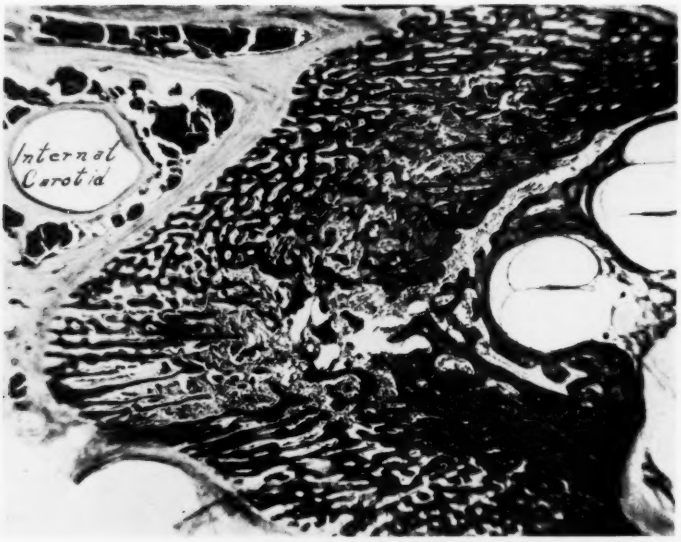

Fig. 5. No. 5454, stillborn, ten lunar months. Negro. Note well developed apex.

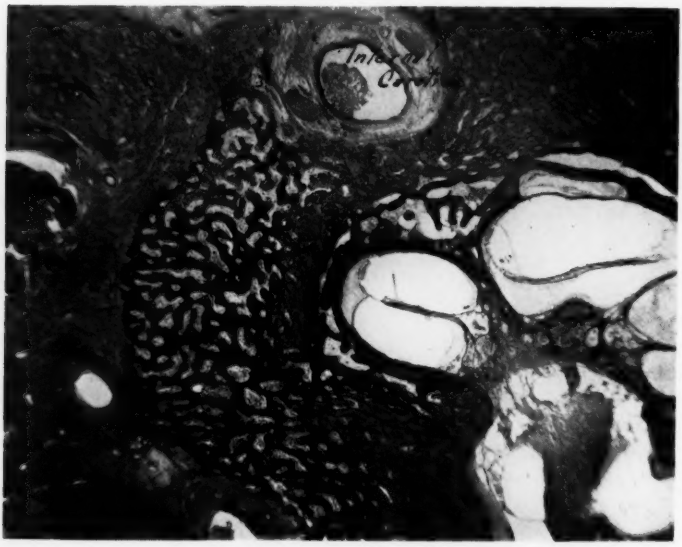

Fig. 6. No. 3937, ten days, full term infant. White. Apex is not so extensively developed as that in Fig. 5.

canals are prominently seen, thus making obvious the relatively large size of the fetal labyrinth.

The first evidence of a petrous apex in our material is illustrated in Fig. 4, of a 6 months premature infant. Here we see anterior to the enchondral bone surrounding the cochlea a well developed layer of lamellated bone. The distance from the lumen of the basal turn of the cochlea in the mid-modiolar section to the anterior osseous boundary is 2 mm.

Great variation in the rate of development of the apex occurs at about the time of birth. This is made evident by measurements found in cases of full term and 10-day infants (Fig. 5). The distance is 7 mm. in No. 5454 for the region described above, as compared with case 3937 (Fig. 6), a 10-day infant, also full term, which measures only 4 mm. A 2-day-old specimen, No. 4324, is 5.5 mm. These wide variations seem to indicate that this part of the skeleton must be studied in relation to the development of the skull and body as a whole.

There is some indication that the presence of middle ear infection may influence the rate of elongation of the apex. A comparison of right and left ears in Case 4232 is a case in point. In this 2 months infant the right, which is heavily infected, measures more than 5 mm. The left, which is essentially normal, is over 7 mm. A second illustration of this is in No. 5426, where the infection was greater on the left than on the right and the length of the apex is over 2 mm. less. Our measurements for the adult averaged 18 mm. The measurements, as stated above, were taken from the most anterior contour of the basal turn (midmodiolar sections) forward to the terminal contour of the apex. Profant reported an average length of 19 mm. for the adult.

So far as we have observed, pneumatization of the apex is a question of anatomic variation. We have no proof that the degree of pneumatization is related to the presence or absence of infection. The apex in specimen 5017 (Fig. 7), a 4-year-old child, is well pneumatized immediately under the gasserian ganglion, then a dense region of hemapoetic marrow occurs and below this another chain of pneumatic cells runs forward. The ear is essentially free from disease, but there are present dense bands of scar tissue in the niche of the oval window.

Specimen 5563 (Fig. 8) could not be included in the table of measurements, as it was cut a little too short. We include it in these observations because of the presence of a large cell in the apex which

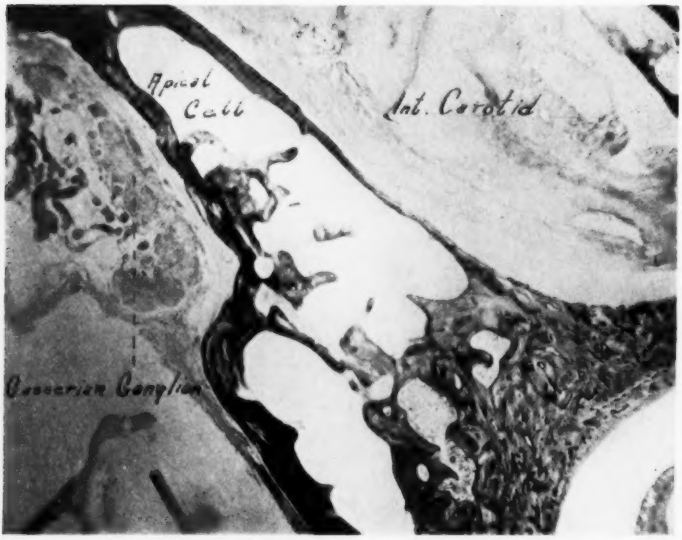

Fig. 7. No. 5017, four years. White female. Well developed and pneumatized apex.

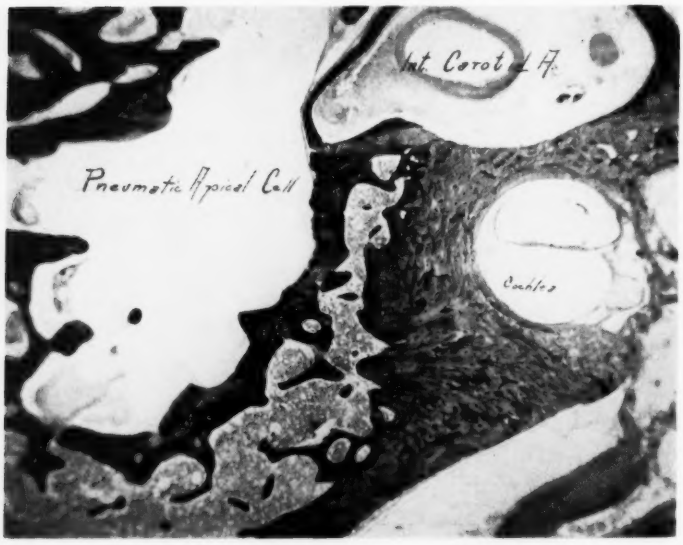

Fig. 8. No. 5563, seven years. White male. Apex well developed and well pneumatized.

communicates with the peritubal cells. The possibility of this condition has heretofore been doubted by some authors.

From our observations we conclude: (1) That the petrous apex or tip first begins to form in the sixth month of fetal life; (2) that pneumatization of the apex may begin as early as four years, long before puberty, but is always more tardy than that of the mastoid; (3) that the apex elongates rapidly up until the fourth year and very slowly thereafter.

TABLE SHOWING RATE OF DEVELOPMENT OF PETROUS TIP

Autopsy No.	Age	Sex	Race	Side	Length	Infection	Pneum.
3887	6 mo. pre.	M.	N.	Left	2 mm.	Slight	
EMC2	7 mo. pre.	?	?	?	2.5 mm.	Present	
3924	4 da. pre.	M.	N.	Rt.	5.4 mm.	Present	
5454	Term	F.	N.	Rt.	7 mm.	Present	
4324	2 days	M.	W.	Left	2 mm.	Slight	
3937	10 days	F.	W.	Left	4 mm.	Present	
4232	2½ mos.	M.	N.	Rt.	6 mm.	Present	
*4330	2 mos.	M.	W.	Rt.	7 mm.	Present	
5426	2 mo.	F.	W.	Rt.	10.5 mm.	Present	
5426	2 mo.	F.	W.	Left	8 mm.	Heavy	
*3967	2 mo.	M.	W.	Left	10 mm.	Heavy	
6012	3 mo.	M.	W.	Rt.	8 mm.	Heavy	
*3947	7 mo.	F.	N.	Left	11 mm.	Heavy	
5017	4 yrs.	F.	W.	Rt.	14 mm.	Scar Tis.	Mixed
*3951	5 yrs.	F.	W.	Rt.	16 mm.	Heavy	Mixed
5115	6 yrs.	M.	W.	Rt.	17 mm.	Heavy	
5115	6 yrs.	M.	W.	Left	17 mm.	Heavy	
5584	6 yrs.	F.	W.	Rt.	15 mm.	None	
*3992	7 yrs.	F.	W.	Rt.	14 mm.	Moderate	Mixed
*3992	7 yrs.	F.	W.	Left	14 mm.	Moderate	Mixed
4280	7 yrs.	F.	W.	Rt.	14 mm.	Heavy	
*3903	8 yrs.	M.	W.	Left	13 mm.	Slight	Mixed
*3956	10½ yrs.	F.	W.	Rt.	16 mm.	Heavy	
5409	15 yrs.	M.	W.	Rt.	15 mm.	None	
5085	31 yrs.	M.	W.	Left	23 mm.	None	
4374	35 yrs.	M.	W.	Rt.	17 mm.	None	
5535	44 yrs.	F.	W.	Rt.	18½ mm.	Slight	
*4098	48 yrs.	F.	N.	Rt.	18 mm.	Scar tissue	
5178	55 yrs.	M.	W.	Rt.	13½ mm.	None	

*These specimens were used in Glick's study, but no measurements were made by him.

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XXXVIII

PRIMARY CHOLESTEATOMA OF THE MASTOID

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The purpose of this communication is: First, to present a case of cholesteatoma of the mastoid without middle ear involvement, and second, to briefly discuss the etiologic mechanisms which may produce this type of mastoid disease.

REPORT OF A CASE

Case 1.—W. R., male, age 29, presented himself for examination because of right-sided temporal headache and discharge from his right ear which had persisted for six weeks. He had had no earache, tinnitus, apparent reduction of hearing, or upper respiratory infection. There was no history of previous ear trouble except that twenty-four years ago a foreign body had been removed from his right canal through a Wilde's incision.

Examination revealed a right-sided postauricular scar. Both drums were mildly retracted, but were otherwise normal. The right canal contained an amount of serious discharge which came from a fistula surrounded by a small amount of granulation tissue in the posterior canal wall. These granulations were infected and proved to be the sole source of the secretion seen in the canal. A probe passed through the fistula entered a cavity in the mastoid bone. There was no mastoid tenderness. Systemic findings were negative. X-ray revealed well pneumatized mastoids with a large area of absorption in the right. Functional tests established slight reduction of hearing in both ears, which proved by inflation to be on a tubal basis.

An antrotomy was performed on the right side, during which the following findings were noted. The cortex was intact. The bony wall showed no evidence of either surgical or infectious destruction, except for a small perforation through its posterior aspect. Removal of the cortex exposed a large cholesteatoma which had destroyed most of the cellular structure of the mastoid and a great part of the dural plate over the posterior and middle fossæ. The exposed sinus and dura were normal. An excellent view of the autrum was obtained. It was found to contain no discharge and was otherwise normal. The cholesteatoma itself was clean and colorless, having the gross appearance of a large pearl. Microscopically large nests of cholesterin crystals were seen, through which endothelial lymphocytes, giant and epithelial cells were distributed. No granulation tissue or pus was encountered throughout the entire mastoid. The patient made an uneventful recovery.

ETIOLOGY OF CHOLESTEATOMA

Cholesteatoma of the mastoid may result from chronic infection, primary congenital growth or a displacement of epithelial cells

into the mastoid bone through growth, trauma or a combination of both. In the case just described no evidence of aural infection, either acute or chronic, was encountered, with the exception of the infected granulation tissue found in the external auditory canal. However, it is unlikely that either the fistula or the granulations had been present for more than the six weeks' period of the aural discharge, so that it is illogical to conclude that the cholesteatoma was secondary to an infectious process in the canal.

The possibility that epithelial tissue was displaced into the mastoid at the time of the removal of the foreign body must be considered. Unfortunately, the otologist who performed the operation is deceased, and no hospital records could be found. However, at the time of antrotomy the cortex and bony canal were intact, not even evidences of bone regeneration being encountered. In the light of these findings it is unlikely that bone work was done during the removal of the foreign body, so that it is difficult to explain how epithelial tissue giving rise to cholesteatoma could have been displaced into the mastoid at this time.

Since it is unlikely that this cholesteatoma resulted from either chronic infection or displaced tissue, primary congenital growth must be considered. Histologically there is apparently no way of distinguishing primary from secondary cholesteatoma. History and examination offer the best methods for differentiation, but the clinical pictures are often as debatable as in the case just described. Teed has recently published a comprehensive monograph with a complete bibliography on cholesteatoma verum tympani. He pointed out that true primary cholesteatoma of the mastoid was very rare, experienced clinicians such as Siebenmann, Politzer and Wittmaack never having seen one. Of all cases of cholesteatoma reported in the literature, Teed has selected but twenty in which growth was probably primary. These cases fall into four groups: 1. Cholesteatoma associated with congenital ear deformity in which atresia of the canal is present; 2. cholesteatoma of the drum membrane; 3. cholesteatoma associated with dermoids, and 4. cholesteatoma of the petrous apex with normal mastoid and middle ear. He believes that these primary epithelial growths result from anomalous functioning of normally placed epidermal cells. These epidermal cells are present in the dorsolateral pole of the tympanum and normally become transformed into epithelial cells. Occasionally, however, the ectodermal quality persists, produces skin, desquamates and forms cholesteatoma. To show why these ectodermal cells are normally found in the tympanum, he presents an extensive review of the phylogeny and ontogeny of the ear.

CONCLUSION

A case of cholesteatoma of the mastoid without middle ear involvement is presented. A critical study of the case leads to the deduction that it is likely a primary cholesteatoma of the mastoid.

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REFERENCE

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XXXIX

PRIMARY ULCERATED INFILTRATIVE TUBERCULOSIS OF THE TONSILS, VELUM AND PHARYNX*

With Report of a Case in Which Healing Followed Treatment With 50 Per Cent Trichloracetic Acid.

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Tubercular infection of the pharyngeal area can manifest itself clinically in four different forms: (1) as tumor-like structures, called tuberculomas, (2) as miliary lesions, (3) as lupus, and (4) as deeply and diffusely infiltrative lesions, which may be referred to as "infiltrative tuberculosis." It is the fourth type with which we are concerned, and in that type as a primary ulcerated infection. By primary we mean the absence of evidence in the history, physical signs and x-ray findings of active tubercular lesions elsewhere in the body which could have produced the lesions in the throat. Although autopsy reports and tuberculin tests show that, in populated areas, from 70 to 97 per cent of adults over fourteen years of age have already been infected with tubercle bacilli some time earlier in life,[1] the lesions of the throat should be considered the primary clinical disease, even though it may occur by way of a blood stream infection coming from some undiscovered latent focus. By ulcerated, we are excluding the so-called "latent tonsillar tuberculosis," namely, that group of extirpated tonsils in which tuberculosis is discovered only on histologic examination and on animal inoculation.

There is no difficulty in distinguishing tuberculomas from the other three types mentioned. Tuberculomas may appear alone or as a part of a lupoid or an infiltrative tubercular infection. However, chronic isolated miliary lesions of the throat, as distinguished from those occurring as a part of an acute miliary tuberculosis of the body, are not so easily differentiated from the diffusely infiltrative type of lesion. The miliary lesions of the throat may not be seen by the physician until the minute, mouse-nibbled ulcers have coalesced and the surrounding tissues appear deeply infiltrated. Such may be the case

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when a hidden focus in the body is sending into the blood stream, at infrequent intervals, small numbers of bacilli which, having reached the tonsils, velum or pharynx, have excited perivascular tubercles and then, by union, a more diffuse infiltration, followed by deep caseation and necrosis. When such isolated lesions occur the large coalescent ulcers may be surrounded by miliary infections which indicate the mode of onset of disease. It will be necessary to classify such lesions as of the fourth type mentioned above, since it is not the endogenous or exogenous origin of the infection but the anatomic appearance which makes the classification. However, the vast majority of miliary lesions of the throat are a part of a widely disseminated body infection, an acute miliary process. In such cases there is no question but that the throat lesions are secondary to a primary, active focus elsewhere in the body, and hence they are not considered as primary in our definition of the term.

The ulcerations occurring in lupus of the pharyngeal area are often confused with those of infiltrative tuberculosis. Lupus is a surface infection, a more or less attenuated form of the disease, while infiltrative tuberculosis is a deep, diffuse, edema-like process, fairly rapid in its progress. Lupus is more apt to be a primary infection, starting in the nose or skin close to the nostril and invading the pharynx, less frequently the larynx; while infiltrative tuberculosis is not so apt to be primary, coming usually from a pulmonary phthisis and involving the larynx most frequently, the pharynx less often and the nose rarely. In lupus, the onset is insidious, in the form of minute, discrete, apple-jelly like nodules which have an indolent tendency to coalesce and form superficial serpiginous ulcers, the ulcers having a tendency to spontaneous healing with extensive scarring of tissue. The secretion in the ulcers is slight and tends to dry in sticky patches. In infiltrative tuberculosis the onset is also insidious but with diffuse, deep, edematous nodules which may become fibrotic and leave small, hard lumps, or more often they become yellowish and slough. The ulcers rarely show any tendency to spontaneous healing and develop firm, irregular edges and deep, dirty-gray craters filled with secondary infection. Lupus is generally painless, dysphagia being the exception and the ulcered surface in the pharynx inclined to be anesthetic and "wooden." The ulcers of infiltrative tuberculosis of the throat are extremely painful, dysphagia being the rule, and the throat is tremendously hyperesthetic.

Only lesions of the palatine tonsils, velum and oropharynx will be discussed, since the case to be presented had lesions limited to this area. While primary lesions of the lips, gums, buccal mucosa, hard

palate, tongue, lingual tonsil, pharyngeal tonsil, nasopharynx and laryngopharynx are also rare and the mode of invasion and onset of disease in these structures just as interesting, space does not permit discussion of these lesions also. The retropharyngeal lymph nodes are situated in the oropharynx and may produce, though very rarely, a tubercular retropharyngeal abscess. The interest in this presentation is centered in primary infection in the mucosa and in the tonsils and not in the draining lymph nodes. Hence this affection is also excluded.

Few cases of primary ulcerated infiltrative tuberculosis in this area of the body have been reported in the literature. It speaks for the rarity of such lesions. Even in cases secondary to pulmonary tuberculosis, the pharyngeal involvements are comparatively rare and encountered mostly in terminal cases. According to Guttmann,[2] about 1 per cent of hospital phthisis cases are complicated by gross lesions of the soft palate. A fair estimate of secondary gross lesions of the pharynx and tonsils was given by Levy[3] in 1910, who reports involvement of the pharynx alone in 2 per cent, the pharynx and tonsils in 1.5 per cent and the faucial tonsils alone in .5 per cent of tubercular patients. A careful search reveals but 56 cases of primary infiltrative tuberculosis of the faucial tonsils, oropharynx or soft palate ever reported in the literature. Where a case was described as distinctly lupoid or secondary in character, it was excluded. However, most cases described in the literature, especially the earlier observations, were not differentiated clearly, both with regard to the form of the lesion and to the lung findings. In many there was no mention made of laboratory evidence. Under the circumstances it is difficult to make an exact list. In many cases pulmonary involvement was noticed soon after the primary involvement of the throat. These cases are included as primary, since there is no reason for doubting that tuberculosis could take a rapid course after primary manifestations appear in the throat. The following is a list of the cases found in the literature:

1875: Isambert, E.: Ann. d. Maladie d' l'Oreille, etc., 1:27.
Boy, 4½ years old. Primary miliary lesions of velum. He appeared scrofulous. Acute miliary tuberculosis with lesions of the velum and pharynx have since been called Isambert's disease, although they do not refer to primary lesions as in this first case.

1876: Frankel, B.: Berl. Klin. Wchnschr., 8:657.
Collected six cases of primary palate lesions. All adults.

1881: Kussner, B.: Deut. med. Wchnschr., p. 277-281.
Collected five cases involving palate and tonsils, all in males between 34 and 56 years of age. Some started with miliary lesions. None developed lung complications when reported. One complicated by lues.

1881: Kessler: Arch. f. Otol., 16:75.
Primary of palate and pharynx involving ear.

1884: Uckermann, N.: Norsk. Magax. f. Lagev., 14:9.
Woman, 35 years. Primary palate lesion. Cured after one month's treatment with 20 per cent carbolic acid in glycerin.

1884. Wroblewski, W.: Int. Central f. Laryng., p. 214.
Reviews 14 cases of pharyngeal involvement, one of which is primary.

1886: Schleicher: Anals. d. la Soc. de Med. d'Auvers, Ferrier.
Male, 30 years. Started in pharynx and involved larynx. Lungs negative throughout.

1887: Lublinski: Int. Central f. Laryng., p. 389.
Primary lesion of right tonsil and uvula.

1888: Kaurin, E.: Int. Central f. Laryng., p. 564.
Girl, 22 years. Tonsils, velum and pharynx. Followed in 3½ months by tuberculosis of lungs.

1889: Lavallee, M.: Quoted by Cornet in Nothnagles Encyclopedia (Eng. Edition, 1904), Vol. on Tuberculosis, p. 129.
Case started in larynx, then pharynx, then tongue. No lung signs after seven years' observation.

1890: Gleitsmann, J. W.: N. Y. Med. J., 52:404-406.
Two cases of pharynx healed with lactic acid. One without lung signs after twelve years. (Reported again in 1901.)

1890: Wohlauer, R.: "Uber Pharunx Tuberculose." (Berlin, g. Shade.)
Thirty-one page book discussing subject. Mentions a case of primary pharyngeal tuberculosis which was followed soon after by tuberculosis of lungs.

1892: Herzog, M.: Cincin. Lancet-Clinic, 28:682-686.
Man, 33 years. Right tonsil and pharynx, ulcers healed with Paquelin cautery. Later developed tuberculosis of larynx. Lungs clear throughout.

1892: Julien, M.: Ann. d. Derm. et Syph., 3:207.
Girl, 26 years. Pharynx and left tonsil. Lungs clear. Presented for discussion of treatment. Lactic acid suggested. Patient was a prostitute who cohabited for six months with a phthisis case.

1892: Koster, H.: Goteborgs Lak-Sallsk., Forh., p. 35-38.
A case of primary tuberculosis of pharynx.

1895: Griffin, E. H.: N. Y. Med. J., 61:209.
Girl, 19 years. Pharynx. Lung infection started one month after onset and died in four months.

1896: Pluder, F.: Arch. f. Laryng., 4:119.
A case of primary tuberculosis of pharynx.

1896: Chappel, W. F.: N. Y. Med. J., 64:377-379.
Woman infected by her two sisters with primary tuberculosis of pharynx. Died soon after with tuberculosis of lungs.

1898: Plicque, Abst. in Laryngoscope, 5:189.
Child, 4½ years. Uvula and pharynx. Rapid and fatal. Quotes Siegert (Jahrbuch f. Kinder-Heilkunde, Vol. 45), who collected twelve similar cases in children—all rapidly fatal.

1899: Barbier, H.: Gazette des Hospit., 72:121-123.
Woman, 32 years. Coalescent miliary ulcer of velum. X-ray therapy not effective. Died a few months later of pulmonary tuberculosis.

1902: Dabney, S. G.: Louisville M. J. Med. and Surg., 9:394-397.
Male, 37 years. Coalescent miliary ulcer of velum. X-ray therapy not effective. Died a few months later of pulmonary tuberculosis.

1904: Newcomb, J. E.: Laryngoscope, 14:423-430.
Male, adult, with primary tuberculous ulcers of pharynx. Treated with lactic acid. Improved.

1917: Horgan, J. B.: J. Laryng., Rhin. and Otol., 31:1-3.
Male, 69 years. Primary miliary tuberculosis of pharynx and larynx. Rapid course and died of purpura hemorrhagica.

1925: Brown, A. E. and Doig, K.: M. J. Australia, 1:86.
Male, 28 years. Started in tonsil and extended to velum and pharynx. Died six weeks after onset with acute miliary tuberculosis.

1927: Kowler: Bull. d'oto., rhin. et laryng., 25:450-453.
(a) Woman, 25 years. Started in tonsils and involved lung one month later. Heliotherapy not effective. Died.

(b) Man, 28 years. Tonsils removed, followed by the ulcer (tuberculous) of velum. Heliotherapy not effective. Lungs later involved. Discontinued treatment.

1928: Horgan, J. B.: J. Laryng. and Otol., 47:338.
Woman, 53 years. Ulcers of left tonsil. Tonsillectomy. Tuberculosis in sections. Pillars and pharynx not involved. Wound healed. Lungs clear.

1930: Dickey, L. B.: Arch. Pediat., 47:90.
Boy, 6½ years. Ulcer of right tonsil. Tonsils removed. Tuberculosis in sections. Well after three years' observation. No lung signs.

1932: Cantonnet-Blanch, P.: Arch. d. Ped. d. Uruguay, 3:273.
Primary of pharynx, treated with galvano-cautery.

1933: Preston, H. G.: Virginia M. J., 60:10-18.
Woman, 27 years. Velum ulcer developed after removal of latent tuberculous tonsils. Later developed syphilis with spontaneous healing of ulcer, in spite of no improvement with all methods of treatment previous to this.

1933: Hamperl, H., and Wallis, K.: Zeit. f. Hals, Nasen u. Ohr. 32:480.
Primary of tonsils. Not reviewed.

1934: Vasiliu: Spitalul, 54:404.
Primary of tonsils. Not reviewed.

1935: Carrara: Littante, 6:369.
Primary bovine of tonsils. Not reviewed.

The relative infrequency of secondary infiltrative tuberculosis of the pharyngeal structures and the extreme rarity of primary lesions proves that these organs are endowed with great resistance to tubercular infection. What this local tissue resistance consists of is yet un-

known. Whether a biochemical or phagocytic or anaphylactic phenomenon creates this immunity is yet to be discovered. It would be of interest to study the processes of invasion and the defensive mechanisms involved, so that a comprehensive understanding may be had of the ways by which, in primary infection, these defenses break down and produce local caseation and necrosis.

In 1889 Cornet[5] had originally demonstrated that by gently rubbing cultures of tubercle bacilli or infected sputum on the buccal mucosa and on the pharyngeal and tonsillar mucosa of guinea pigs without abrading the surface, no lesion will occur locally; however, bacilli will be discovered later in the adjacent lymph nodes. In case the mucous surface is abraded, ulceration will usually occur first at the site of abrasion. Since then, both experimentally and clinically, the same phenomenon has been noticed in the alimentary canal as well.[6] That invasion through the alveolar lung tissue can take place also appears possible, but experimental evidence seems to prove that few organisms, when inhaled, reach further than the bifurcation of the bronchi and that invasion of the lungs is usually through hematogenous channels.[6] The common modes of invasion of the body are as follows: (a) the inhalation of dust or droplet laden bacilli which mix with the mucus of the mouth, throat and trachea; (b) the ingestion of infected food, principally milk; (c) the ingestion of infected mucus from hand to mouth through contaminated fingers, towels, clothing, toys, and the like; and (d) the ingestion of mucus from mouth to mouth as in kissing. Invasion through the abraded skin is not very common and invasion through the intact skin, although produced experimentally, does not appear of importance clinically. That placental infection ever takes place is still a moot question. Since the presence of either tubercle bacilli or of tubercles in the organs of the newborn is still to be demonstrated, it seems safe to assume that all tuberculosis in children and adults is a postnatal infection.

There are two strains of tubercle bacilli that are important to man: the bovine type and the human type. The bovine type is almost exclusively carried in milk and is of primary importance as an infection only in children. While the bovine type is found in 10 per cent of children and usually produces milder forms of the disease, such as glandular, bone, joint and skin conditions, the human type is the cause of about 99 per cent of adult tubercular lesions and of 90 per cent of the more serious forms in children.[4a] Hence contaminated milk does not play nearly as important a role as contaminated sputum.

Once the bacilli enter the submucosa they travel to other parts of the body through the blood and lymph streams. They are either taken up by the neutrophilic leucocytes and are thus distributed through the blood stream to other parts of the body or they travel rather slowly in the tissue lymph spaces and in the current of the lymph ducts when these are reached. Histologic tubercles may form in the submucosa and remain dormant for some time, but as a rule they appear most abundant in the adjacent lymph nodes. It is not requisite that the bacilli develop to any extent or at all at the site of invasion.

The tonsillar tissue is one of the common portals of entry of the tubercle bacillus. For many reasons, it is a more common portal of entry than the velum or pharynx. Unlike the tonsils there are no crypts in the pharyngeal mucosa in which the bacilli may lodge in numbers. The abundant mucus holding the organisms away from the surface of the pharyngeal mucosa, and the act of swallowing and coughing which sends the mucus constantly to and fro, has a tendency to wash the bacilli away before they can gain a foothold. The bactericidal action of the mucus must play some part in getting rid of bacilli before entrance is affected. Should any organisms gain entrance through the intact pharyngeal wall, the lymph follicles in the submucous tissue could nest them just as readily as those in the tonsils, but the rich lymphatic drainage of this area carries most of them away to other more adaptable parts.

The bacilli may lodge in the tonsils, forming microscopic tubercles, and give no clinical evidences of tubercular disease. A small focus in the tonsils may produce extensive involvement of the lymph nodes and vice versa. The gross appearance of this so-called "latent tonsillar tuberculosis" shows no different picture from that in ordinary chronic inflammation. Although acute exacerbations do occur, ulceration rarely occurs. In fact, latent tuberculosis of the tonsils, being chronic in type, leads to fibrosis and shrinkage, so that the tonsils are often small and atrophic and are not suspected of specific infection. The incidence of latent tuberculosis of removed tonsils, where no clinical evidence of tubercular disease is manifest in the glands or elsewhere in the body, ranges, according to many investigations made up to 1934,[7] from 0.5 per cent to 8.0 per cent. When the cervical glands are involved, Mitchell[8] found evidence of latent tonsillar tuberculosis to be 37.5 per cent. When the lungs are involved, Wood's table[9] of seven observations made up to 1905 gives 69 per cent average, and an estimate made in 1934 by Newhart, Cohen and Van Winkle[7] makes it 48 per cent. The incidence of

latent tuberculosis of the pharyngeal and palatine tissue has never been taken. It would be of interest to take biopsies of the lymph follicles of the pharyngeal wall in cases of removed latent tubercular tonsils. The defensive mechanisms in the pharynx being greater than those of the tonsils, the incidence would probably be smaller. Secondary clinical tuberculosis of the tonsils is rare, when compared with secondary latent tuberculosis of these organs. While Wood estimated 69 per cent of latent tonsillar tuberculosis in phthisis patients, Levy found clinical tonsillar tuberculosis in only 2 per cent of such cases.

Tubercle bacilli may remain latent in the lymph glands of human beings without producing gross or even microscopic changes for many years, in fact, for the natural life of the individual. Bartel[10] refers to this condition as "latent lymphoid tuberculosis." Even in calcified and fibroid glands, virulent bacilli have been recovered where not even microscopic evidence of infection could be found.[11] While under ordinary circumstances these latent foci serve to immunize the fairly resistant individual against reinfection, at any time when the resistance is at a low ebb, as when some intercurrent affection takes place, the bacilli may invade the blood or lymph stream and thus be carried to some vital organ. The most vital organ is the lung, but it is possible that a chronic miliary auto-infection may leave no definite clinical signs in the lung and manifest itself in the pharyngeal structure instead.

Knowing that there are latent foci in the body, we can ask ourselves: When primary ulcerative tubercular lesions appear in the throat, is it due to exogenous reinfection or is it an endogenous metastasis from a dormant lesion? The exposure of the surface area of the throat to outside contact and its susceptibility to abrasion speaks well for exogenous reinfection. But, as has been shown by Koch's phenomenon[12] and by other experiments of recent workers,[4b] once an individual has successfully resisted a primary tubercular infection it is exceedingly difficult for him to receive an exogenous reinfection. If the reinfecting dose is small, perfect resistance takes place. However, this immunity succumbs to a sufficiently virulent and massive dose.[13] That endogenous reinfection is just as likely as reinfection by the exogenous route is elicited from the fact that a single massive dose is usually not the rule in exogenous invasion, hence the repeated reinfection from within, coming at a time when the body resistance is lowered, must be considered just as frequent an occurrence. Newhart, Cohen and Van Winkle[8] are of the opinion that when pulmonary tuberculosis exists, hematogenous infection of the tonsils is the exception rather than the rule. Here massive doses coming from the

sputum of the lungs can produce exogenous reinfection of the tonsils more readily than in primary reinfection of these organs. It may be mentioned at this point that a bovine infection in childhood may immunize the individual from serious reinfection with the human bacillus.[14] However, when reinfection with the human bacillus occurs in adults, as is the rule, it is not due to an endogenous bovine organism, since it has not yet been proven that mutation of the bovine to the human type can take place in the single span of life of an individual.[15]

The ways by which, in primary infection of the tonsils, velum and pharynx, the local defenses break down and ulceration ensues appear as follows: Local ulceration may occur through endogenous or exogenous reinfection on an abraded surface. The abrasion may be mechanical, such as caused by toothbrush bristles, rough foods, swabbing with applicators and the like; or it may be of infectious origin, such as aphthous ulcers or vincent's, thrush and luetic lesions. Repeated severe catarrhal infection of the nose and throat may undermine the epithelium and cause abrasions. Such mechanical or infectious abrasions may either light up a local latent tubercular infection or be infected by tubercular sputum or food or blood stream derivatives where no latent infection existed. Where no local abrasion or undermined tissue exists it may be reasoned that a massive dose or even a small virulent dose, reaching the throat by exogenous or endogenous routes, can produce local necrosis similar to a strong tuberculin reaction in a highly immunized individual. Baldwin[16] states that many local reactions are due to this allergic response and quotes Koch's experiments with tuberculin in which a dose sufficient to kill an animal can produce extensive necrosis of the skin in man in the region of the injected spot. It is thus possible that a large dose or a virulent small dose may produce the same local reaction in the throat of a highly sensitized individual.

The spread of tubercular ulcers from one area of the throat to another may be caused by operative trauma or by contact. Preston[17] describes a case in which ulcers of the pharynx and velum followed the removal of latent tubercular tonsils. It is most likely that operative trauma to the palatine epithelium caused it to be infected by organisms expressed from the tonsils during their removal. In the case I am going to describe, ulceration of the palate and pharynx were present with those of the tonsils. When the ulcers were first treated and considerable improvement followed, the removal of the tonsils helped to hasten the cure. Here the elimination of areas of contact reduced the number of secondary foci. The contact of the

ulcers with the surrounding tissues induces undermining of this tissue by the secondary infection present in the ulcers and thus the tubercular infection takes hold.

Space does not permit discussion of the usual symptoms of primary tubercular ulceration of the throat. It is important, however, to stress the insidious onset. Before ulceration, areas of infection may exist for months without producing pain, and patients may not consult the physician until the lesions are far advanced. When first seen by the physician, the picture may be misleading, there being no history of tuberculosis of the lungs nor evidence of pulmonary signs or symptoms except emaciation. A secondary infection of Vincent's angina or thrush is misleading. A history of syphilis or a positive blood test for this disease may lead one to suspect tertiary luetic lesions. The two diseases may be associated in the same lesion. The case reported by Lack[18] of a female, aged 25 years, in which tubercle like nodules were found in the right tonsil but in which the administration of potassium iodide cured the lesion, must be looked on as one of syphilis associated with tuberculosis. The tuberculin test when presenting focal reactions is pathognomonic, but with only local and general reactions it is of little value. A biopsy of the lesions is essential. Although the presence of tubercles does not belong exclusively to tuberculosis, the demonstration of tubercle bacilli, either on the slides and on cultures or on animal inoculation, is conclusive evidence. However, organisms cannot always be demonstrated. In such cases the characteristic gross lesion and histologic picture should be taken as sufficient proof when no other misleading facts prevail.

There remains but a discussion of the treatment before the case is presented. This is dealt with somewhat in detail because of the unusual procedure undertaken and the remarkable result obtained. The usual treatment advocated for tubercular ulcers of the tonsils and pharynx consists in the use of cleansing irrigations, astringent and iodin powders and general hygiene, the cautious use of tuberculin injections in well selected cases, the curetting of the craters, the cauterizing of the ulcers, principally with the galvanocautery, the Paquelin cautery and the fulguration point, or with chemical caustics such as 75 per cent lactic acid, pure chromic acid and formalin, the coagulation of infiltrated areas with the high frequency current, the excision of small ulcers with the electrosurgery knife[19] and the use of ultra-violet rays, direct sunlight and the x-ray. Although the extirpation of latent tubercular tonsils is advocated, it is the general concensus of opinion that the removal of clinical tubercular tonsils

should be undertaken with great caution, especially when an active focus elsewhere in the body is suspected.[3 4 7]

In none of the cases reported in the literature was trichloracetic acid used to cauterize the ulcers of the throat. In 1932 Davis[20] reported the use of trichloracetic acid in full strength to cauterize ulcers of the larynx. He used it over a period of six years on 6,000 patients at the General Hospital Laryngeal Clinic of Los Angeles with excellent results. Mention is made in his article of D. Bonain of Brest, who used trichloracetic acid in tubercular ulcers of the larynx in 1922, and also of Okouneff of Petrograd, who used the acid in the treatment of ulcers of the larynx as far back as 1901[21] with good results. Okouneff advocated a wider use of this treatment in ulcers other than in the larynx. The interesting feature in the case I am going to present is the remarkable recovery after the use of trichloracetic acid when many other methods of treatment failed.

A detailed report of the history, findings and treatment of this patient follows:

REPORT OF A CASE

CASE 1.—R. B., 33 years of age, Italian, single, a waiter, was first seen by me on January 27, 1936. He complained of pain on swallowing, which started insidiously six months before. The pain increased gradually and became so severe that for several days before he was seen by me he could hardly swallow his food. He lost 25 pounds since the onset of dysphagia. There was no history of cough, fever or night sweats, but he did complain of frequent attacks of "colds" and of nasal obstruction and of a constant postnasal mucous discharge of several years' duration. The voice was clear and had always been clear before and since the onset of illness. The patient had always enjoyed fairly good health, could not remember any illnesses in childhood, had never been acutely sick in his adult life, and had periodic examinations of his heart and lungs, which were always found in a healthy condition. Ten years before, he had been treated for a gonorrheal infection of the genital organ and had been pronounced "cured." At that time a blood Wassermann was taken and found negative. There was no history of tuberculosis in the family and no intimate contact with tuberculous individuals. He was in the habit of smoking about six cigaretes a day but did not indulge in alcohol. Since the onset of illness no local treatment was given by a physician. However, during this period the lungs were examined several times clinically by three general practitioners and were found nonpathognomonic. The patient had been using various cleansing gargles that were prescribed, but without relief.

On examining his throat extensive dirty-gray ulcers with punched-out verrucous margins were seen on both tonsils, anterior and posterior pillars, the soft palate and uvula and posterior pharyngeal wall. I could count eleven ulcers of various sizes and shapes. Those of the tonsils were deep and surrounded by large masses of infiltration, those on the uvula and soft palate were quite superficial while those on the pharyngeal wall were fairly deep and surrounded by red edematous nodules. A dry mucus crust covered an ulcer reaching towards the nasopharynx.

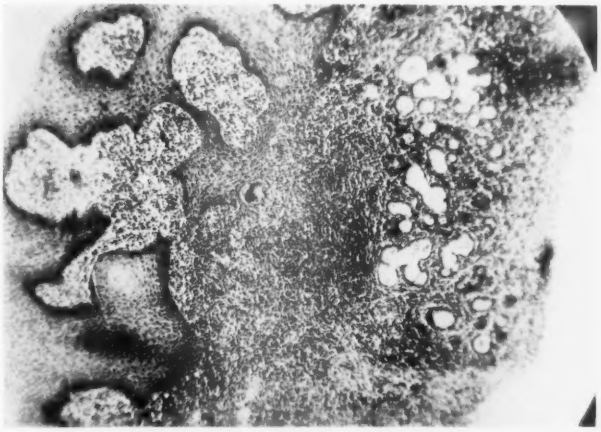

Fig. 1. Section from margin of ulcer of pharynx. Low power. Tubercle with single giant cell forming just beneath the squamous epithelium. No caseation.

When the crust was removed, no evidence of infiltration or ulceration in the nasopharynx could be found. Neither did the ulceration extend down to the hypopharynx. The larynx, epiglottis and base of tongue were not involved. Nor did the nose show evidence of lesions. There was, however, coming from the nose, a chronic, abundant postnasal mucus discharge which could not be definitely localized to any one group of sinuses. This was no doubt irritating the pharyngeal ulcers. X-rays of the sinuses showed a generalized thickening of all mucous membranes with no localized pus pocket. Diagnostic irrigations of the antrums and sphenoid sinuses produced a clear return of fluid. The ears appeared normal. The tongue was heavily coated and the teeth and gums covered with a foul, grayish film. The cervical glands were moderately enlarged, discrete and tender, especially on the right side. Smears taken from the ulcers showed the presence of Vincent angina organisms. Nothing could be elicited from the history or physical signs to indicate the mode of onset of the disease.

Local cleansing irrigations and the use of astringent and iodine powders eliminated much of the secondary infection, but the ulcers persisted. From the first day of treatment the patient experienced considerable relief from dysphagia, showing that most of the pain came from the secondary infection. On the third day of treatment biopsies were taken from the edges of the ulcers. On February 11, 1936, these were reported: "Tuberculous granuloma. Sections show irregular hypertrophy of squamous epithelium and diffuse inflammatory infiltration composed largely of lymphocytes and monocytes, involving the entire sections. In several places there are seen typical tubercles, some of them with giant cells." (Fig. 1.) The sections were not stained for tubercle bacilli. Other biopsies were taken and sent to the laboratory for isolation of the organism. With the Ziehl-Nielson stain these sec-

Fig. 2. Section of tonsil. Low power. Large tubercle with many giant cells and areas of necrosis. Surface ulcerated. Epitheloid cells scattered throughout and the area surrounded by a ring of lymphoid cells except where the surface is ulcerated.

tions showed the presence of tubercle bacilli and, on guinea pig inoculation, the human type was isolated.

The number and size of the ulcers in the tonsils and pharynx and the extent of infiltration was so great that to curette the craters and to cauterize the ulcers with electro-cauteries was impractical because it involved too much tissue. The case was not considered suitable for tuberculin therapy, a method of treatment which is not often successful and is dangerous since it may light up the process. The extensive infiltration and ulceration required some form of medication that would cover the area diffusely. Lactic acid, 75 per cent, was applied for several days, but with no success. Then trichloracetic acid, 50 per cent, was used empirically and swabbed over the whole area of involvement. A stronger solution of the acid was considered too escharotic. With 50 per cent trichloracetic acid the tissues blanched but were not cauterized. The blanched appearance disappeared in 24 hours. The ulcers healed rapidly and the infiltrations gradually disappeared. An attempt to remove some of the persistent infiltrations by the use of electro-coagulation produced more destruction of tissue than necessary. It was therefore abandoned for the slower but safer process of swabbing with the acid. The treatment was given every second or third day for three months. At that time the ulcers had completely disappeared and but a few infiltrations remained.

On April 16, 1936, the tonsils were removed under local anesthesia at Presbyterian Hospital. The specimens were examined in the hospital laboratory and reported as follows: "Sections show giant cells of the Langhans type distributed in the follicles. In some regions there is an infiltration of large cells having large slight-stain-

ing cytoplasm. These epithelioid cells crowd out the tonsillar tissue. A Ziehl-Nielson stain shows scattered acid-fast organisms. Diagnosis: Tuberculosis of tonsils. (Fig. 2). The patient made an uneventful recovery and has been observed by me for the last eight months. Upon further application of trichloracetic acid, 50 per cent, for one month, the few remaining infiltrations of the pharynx had completely disappeared and the swelling and tenderness of the cervical glands had completely subsided. X-rays of the chest, taken on February 14, 1936, and December 17, 1936, revealed the following: "The lung markings show no evidence of active acid-fast pulmonic involvement nor of foci of infiltration or consolidation. There are, however, numerous calcified nodes opposite both hila and several calcified tubercles near the right base." For the last seven months the patient has shown no evidence of recurrence of lesions. He has gained weight and appears in perfect health. Except for a few thin scars on the posterior pharyngeal wall, there are no signs of the former lesions. Attention to the nose has cleared most of the postnasal discharge so that the discomfort from that quarter is slight.

SUMMARY

Diffuse infiltrative tuberculosis is distinguished from lupoid, acute miliary and latent forms.

The rarity of pharyngeal lesions, even when secondary to pulmonary tuberculosis, is reported. An attempt is made to give a complete list of all primary cases in the literature.

A study is made of the modes of invasion of the body and the local defenses of the pharyngeal tissues to resist invasion. The reinfection of the pharyngeal organs is discussed with regard to the exogenous or endogenous routes. And finally the ways are mentioned by which, in primary infection, the local defenses break down and ulceration ensues.

The difficulties of diagnosis are mentioned and a summary of the usual treatment follows.

A case is reported of primary infiltrative tuberculosis of the tonsils, velum and palate which was healed by the use of 50 per cent trichloracetic acid.

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XL

AN OBJECTIVE STUDY OF THE COMPARATIVE NUMBER OF SPEECH SOUNDS SPOKEN PER MINUTE BY THE DEAF AND THE NORMAL*

CHARLES H. VOELKER, M.A.

HANOVER, N. H.

Aristotle did not believe that the deaf could learn to talk, and it was not until 1692 that speech was taught to the deaf, when Ammon described his pedagogy which was to be of such wide moment.[1] The beginnings of the teaching of speech to the deaf was largely the result of the work of a Britisher, Arnold.[2] Now speech is taught to practically all deaf children in this country. Yet recently Shaw said, in writing about deaf pupils who were being taught to speak, "The first and most striking common characteristic of the speech of these ten subjects was the lack of it."[3] It is possible that one factor which may have led Shaw to make this statement before admittedly describing the several defects in their utterance, might be the slowness of speech described qualitatively by Hudgins.[4]

In this paper it is undertaken to describe this slowness quantitatively. In a previous paper an analysis, in terms of words per minute, was made.[5] This research has since been considered as giving an inadequate picture. It could not take into account the elisions of sounds through assimilation and defectiveness, nor the added and altered phonetic elements that should have had no place in the passage. For example, deaf children are apt to say what might be interpreted as "Gooda mornen" for "Good Morning," etc. The immediate outgrowth of the above study of word frequency was to begin anew and analyze the speech of the deaf by counting each and every sound and computing their average frequency in any minute. It was felt that what they said instead of what they should have said was the only consideration within the province of any study of deaf speech rate. This paper, then, attempts to derive an index for the rate of utterance of the deaf based on the number of speech sounds spoken per minute.

The same apparatus set-up was adaptable for this study and is described in the earlier paper and elsewhere.[6] Briefly, deaf children

*From the Speech Clinic of Dartmouth College, Hanover, N. H.

were selected at their optimum of speech education. Their speech was recorded on an audio-record,[7] from which phonetic transcriptions were made. In the case of off-color phonetic quality great care was taken only to determine the number of sounds uttered; the method being the same as that so commonly used in analyzing normal speech —counting the loudness peaks: one peak per sound, e. g., one *b* sound in *cabbage*, two in *cab-boy*.

Simultaneously a visuo-record was made on a film which could be measured in such a way that its linear or running length could be interpreted temporally.[8] The number of sounds and the total time derived from this film of familiar sentences gave by computation an index: the average number of sounds spoken per minute. Ninety-eight deaf cases were used and compared to a control group of thirteen made up of normal hearing children the same age as the deafened, and for the rest, the hearing teachers of the deaf cases.[9]

RESULTS

The criteria of judgment was established by the control group. The normal hearing group spoke at an average rate of 469.1 speech sounds per minute (mean 458.4). The most rapid utterance recorded was 586.2 phonetic elements per minute, which was in contrast to the individual who spoke the least rapidly; his rate was 376.2 sounds per minute. The difference between the most rapid and the slowest speaker, therefore, was 210.0 sounds per minute. The fastest speaker spoke 117.1 sounds per minute faster than average. The most deliberate utterance was 92.9 sounds per minute slower than the average. Table 1 shows the rate, in speech sounds spoken per minute, for the normal hearing control group.

TABLE 1

RATE OF THE INDIVIDUAL'S SPEECH SOUND ARTICULATORY CO-ORDINATION FOR THE NORMAL HEARING CONTROL GROUP

Case Number	Number of Phonetic Elements per Minute	Case Number	Number of Phonetic Elements per Minute
1	586.2	3	453.0
13	583.2	2	411.6
9	561.6	6	403.8
7	520.2	5	389.4
12	514.2	4	379.8
11	461.6	8	376.2
10	458.4		

In comparison to the control group, the deaf spoke at an average rate of 209.8 sounds per minute (mean 192.9). The most rapid

utterance was 405.6 phonetic sounds per minute, and the slowest 79.8 elements per minute. The derived difference between the fastest and slowest utterance was 325.8 sounds per minute. The most rapid speaker spoke 195.8 sounds more than average. The least rapid enunciation was 130.0 sounds per minute slower than average. Table 2 gives the phonetic co-ordination rate for each deaf subject.

TABLE II

RATE OF THE INDIVIDUAL CASE'S SPEECH SOUND ARTICULATORY CO-ORDINATION FOR THE DEAF GROUP AT THE OPTIMUM OF SPEECH PROGRESS

Case Number	Number of Phonetic Elements per Minute	Case Number	Number of Phonetic Elements per Minute
75	405.6	32	192.6
31	398.4	20	192.6
85	372.0	56	192.6
94	331.8	36	192.0
66	300.6	11	189.6
3	295.2	13	187.8
68	286.2	37	185.4
77	281.4	93	185.4
18	274.8	4	184.8
19	274.2	49	183.6
15	278.4	64	183.6
98	268.2	83	182.4
73	256.2	71	182.4
2	252.0	47	180.6
74	251.4	48	178.2
90	250.8	62	177.6
79	249.6	5	177.6
26	245.4	53	177.0
80	244.2	59	177.0
24	244.2	29	177.0
21	242.2	17	175.2
45	241.2	12	172.8
6	235.8	67	172.8
61	235.6	43	171.6
50	231.6	16	171.6
1	228.0	35	168.6
92	226.8	23	168.6
60	225.0	65	168.0
7	225.0	63	166.8
57	223.2	28	164.4
38	222.6	82	160.8
27	222.6	58	160.8
91	219.6	70	157.8
14	219.0	33	156.0
51	216.0	86	154.8
95	210.0	72	151.2

TABLE II—Continued

Case Number	Number of Phonetic Elements per Minute	Case Number	Number of Phonetic Elements per Minute
81	208.8	89	148.2
87	208.2	22	147.0
84	208.2	10	143.4
30	206.4	41	143.4
46	202.8	78	141.0
8	199.8	40	141.0
9	199.9	69	139.8
52	199.2	88	133.8
97	198.6	54	131.4
42	198.0	34	117.6
96	196.8	76	117.0
55	195.6	25	97.8
39	193.2	44	79.8

From the point of view of mean rate, the deaf are subnormal by 265.5 phonetic elements per minute, or in lieu of the average, 259.3. The fastest normal subject spoke faster than the fastest of the deaf by 180.6 sounds per minute. The slowest deaf speaker was retarded 296.4 speech sounds per minute below the slowest hearing subject. The difference between the fastest and slowest speaker for each group was 115.8 sounds per minute greater for the deaf. The fastest speaking subject was above average by 78.7 sounds more for the deaf than normal, and the slowest, 39.1 sounds per minute below average more for the deaf group than in the control group. The most rapid speaking deaf individual was 63.5 sounds per minute below normal average rate and 29.4 sounds per minute faster than the lower normal threshold. The fastest normal rate was 506.4 speech sounds per minute faster than the slowest deaf subject's speed. This subject was 389.3 sounds per minute slower than what normally is average. This is demonstrated by Table 3.

TABLE III

COMPARISON OF THE PHONETIC CO-ORDINATIONS OF NORMAL AND DEAF GROUPS

	Normal Hearing	Deaf
Mean	458.4	192.9
Average	461.1	209.8
Fastest	586.2	405.6
Slowest	376.2	79.8
Difference: Fastest and slowest	210.0	325.8
Difference: Fastest and average	117.1	195.8
Difference: Slowest and average	92.9	130.0

CONCLUSIONS

The retardation of the fastest deaf speaker in comparison to the fastest normal was 64.4 per cent, and the slowest deaf speech was hardly more than a fourth as rapid as the slowest of that which is normal.

The lack of attention to the problem of rate of utterance in the phonetic method of teaching speech in deaf oralism is obvious upon examination of the dispersion of deaf subjects in regard to speed of utterance. This greater spread of 115.8 phonetic elements per minute represents an abnormally large scatter by 64.4 per cent.

The deaf, therefore, spoke on the average 44.7 per cent slower than normal, and would have to speed up their speaking by 79.3 per cent to come within the normal limits. This is not an unreasonable advance in rate, since more than 3 per cent (within the P. E.) spoke as fast as, or faster, than a normal rate, and one of those three cases spoke 10.4 per cent faster than the most deliberate normal speech, which is to say that it was more rapid than 30.7 per cent of the control group.

SUMMARY

A laboratory investigation of visual and auditory recordings of deaf subjects at the end of concentrated speech training were compared to those of a normal hearing control group in order to obtain a comparative estimate of their phonetic co-ordinations. It was found that the deaf were retarded 44.7 per cent. The present lack of attention to normal rate of phonetic pronouncement should be altered and the advisability of deaf oral pedagogy undertaking to emphasize more rapid co-ordinations was demonstrated by the 3 per cent in the deaf group who, in regard to rate, spoke normally.

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XLI

POSTAURICULAR FISTULA*

REA E. ASHLEY, M.D.

SAN FRANCISCO

The process of healing in mastoid wounds does not always pursue to a successful termination the uneventful course which we so greatly desire and so highly appreciate. The healing may be rapid immediately following the operation, but frequently lags behind more and more each succeeding week until the surgeon may become fearful that the wound will never heal. While the great majority of such wounds do ultimately heal, a certain small percentage form a permanent fistula, which either remains open into the mastoid cavity, or is closed by nonresistant scar tissue or a thin, poorly nourished, layer of epithelium. Such wounds not only form unsightly cavities behind the ear, but also are prone to break down and become secondarily infected.

During the early years of mastoid operations the desire was to provide a permanent postauricular opening, which, although disfiguring, was considered essential. Zoufal considered it all important in his description of the first mastoid operations by the radical method. The principle of the operation at that time was to allow epidermization of the cavity to take place from behind the ear, rather than by the present day method of immediate closure of the posterior wound, and subsequent epithelization of the cavity through splitting of the membranous canal. Before adoption of this improved technic fistulæ were the rule, but since then they have become comparatively rare.

Many methods for closing persistent fistulæ following radical mastoid operations have been described, but little appears in the literature pertaining to the closing of postauricular fistulæ following simple mastoid operations. While these also are somewhat rare, they do occur with sufficient frequency, and their correction is sufficiently difficult to warrant our consideration. The method described here has been used successfully in closing fistulæ following both the radical and the simple operation.

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*Presented before the meeting of the Western Section of the American Laryngological, Rhinological and Otological Society, San Diego, January 30-31, 1937.

Fig. 1

Barnhill[1] describes a spontaneous fistula which occurs through erosion of the cortex and soft tissues overlying the mastoid. This type follows acute or chronic infection of the mastoid cells and requires immediate operation.

Fistulæ following mastoid operation usually are of one of three types. The first type presents a firm scar closing both upper and lower margins of the original operative incision, while at a point a little above the center of the cicatrix and corresponding with the situation of the antrum, an opening presents which leads by a fistulous tract inward, upward and forward toward the posterior root of the zygoma.

The opening in the center of the cicatrix, which is surrounded by glossy, shiny, red skin or scar tissue, may be round or tubular in character, nearly devoid of granulations, and apparently beginning to epithelize. A probe introduced may or may not reveal the presence of uncovered bone.

The second type of fistula may demonstrate itself merely as an opening filled with pouting granulations, which, if followed to its termination, usually reveals a sequestrum or an overlooked poorly draining infected mastoid cell.

The third type presents a broad, thin epidermal scar of pale red color which has sunken into the mastoid cavity and has become firmly adherent to the bone throughout its extent. So few granulations have been produced by the bone, and so scanty has been the regeneration of tissue, that the large crater created by the operation remains unaltered in shape and size; and where the bulging convexity of the mastoid process formerly existed there remains now only a deep depression. Down the sides of this pit epidermis has crept in a thin inverting layer, and adheres so closely to the margins and walls of the bony cavity that all its irregularities can be plainly seen and felt. The epithelization does not progress to complete investment of the depression in the mastoid, but is arrested in its deepest portion, leaving either a persistent area of suppuration or a thin layer of scar tissue. This raw spot, or unepithelized area at the apex of the crater, is always very retractable, and at times defies all attempts at epidermization. Such a large excavation in the mastoid region, covered only by thin reddish epidermal scar tissue, constitutes not only an unsightly deformity but an annoying and sometimes painful condition as well.

Mastoid infection which accompanies chronic suppurative otitis, when not eradicated by surgical intervention, is controlled by nature through the formation of granulation tissue within the mastoid cells and cavities. These granulations subsequently undergo organization and osseous changes resulting in osteosclerosis or an eburnized mastoid. With sclerosis there occurs obliteration of the haversian systems with their blood supply and lymphatic drainage. Bone so produced is notoriously liable to chronic infections of low virulence.

An operated mastoid also normally heals by granulation and becomes partly filled with bone. This bone is always of the osteosclerotic type and is especially prone to re-infection. Any factor which interferes with the completion of this orderly normal method of repair may give rise to a fistula.

PREDISPOSING FACTORS

Among the predisposing factors responsible for fistulæ following simple mastoid operation may be mentioned:

1. The general, personal and family clinical history of the patient; the presence of tuberculosis, lues, diabetes, scarlet fever, and many of the blood dyscrasias, favor the formation of fistulæ.

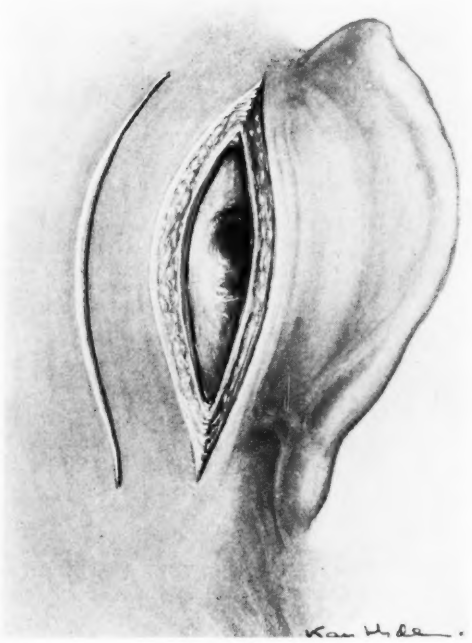

Fig. 2

2. Very pneumatic mastoids where large cavities result from the extensive removal of cortex and mastoid cells. In many such mastoids the cells extend into inaccessible regions where removal is impractical or impossible. These usually follow one of three courses: some go on to uneventful recovery, others require subsequent surgical revision, and the remainder develop fistulæ.

3. Haphazard closing of wound, such as the use of skin clips without careful approximation of the deeper tissues, is a frequent etiologic factor.

4. Errors in after treatment are most important. Packing over too long a period of time with its consequent destruction of healthy granulations; or, conversely, allowing the skin wound to close before healthy granulations have at least partially filled the cavity. How frequently, when called upon to reopen an old mastoid, do we find a large cavity beneath the healed skin which has not granulated and which communicates freely through the antrum, aditus and middle

ear to the eustachian tube, thus favoring reinfection from the nasopharynx. Such conditions certainly are conducive to the development of a fistula.

5. The unavoidable increase of nonresistant scar tissue resulting from multiple operations, due to reinfections of the mastoid cavity, is frequently a contributing factor.

Many fistulæ close spontaneously following complete mastoid revision operations in which previously overlooked mastoid cells are exenterated, and where unhealthy granulations or pieces of dead bone are removed.

As already stated, however, there is a certain small percentage of fistulæ which do not yield to this simple treatment. A discussion of the methods of correcting these persistent fistulæ is the subject of this paper.

Both Popper and McNichols advocate the use of periosteal flaps to cover denuded bone following radical mastoid operations. This procedure, they believe, tends to prevent fistula formation.

Popper[2] advises large horseshoe shaped periosteal flaps, cut from behind the mastoid and attached to the postauricular fold, to line radical mastoid cavities.

McNichols[3] uses similar flaps cut from the temporal region and from the postmeatal wall, to line these cavities.

Dixon[4] describes a new mastoid incision which parallels the posterior border of the external ear when it is held flat against the side of the head. He believes that such an incision heals much more rapidly than the orthodox incision, and that the danger of fistula formation is overcome.

Following radical mastoid operations, breaking down of the postauricular wound, delayed healing, and indications that permanent closure is not likely to occur, can usually be determined early in the postoperative care. This, I believe, is the time to perform the plastic operation—it will hasten convalescence and may forestall fistula formation.

The Mosetig-Moorhof[5] operation utilizes a flap of epidermis cut from below the fistula, turned upward into the fistula, with the epithelial side toward the cavity.

Heine[6] takes the flap from immediately behind the opening, because he believes this gives a better chance of re-establishing nutrition.

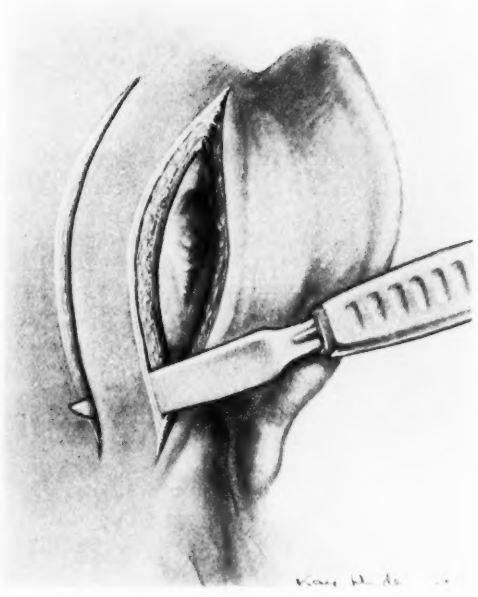

Fig. 3

The Trautmann[7] operation uses the epidermis on either side of the fistula, turning two flaps inward and suturing them together to close the defect. By undermining the skin from which the flaps were cut the fistula edges can be drawn together.

Beck's[8] method is the same as Trautmann's, except that larger flaps are cut and a secondary posterior incision is made to relieve tension.

Goldstein[9] freshens the edges of the fistula and pulls them together by undermining the skin and relieving the tension by a short secondary incision parallel to the long axis of the fistula.

Kerrison's[10] method of closure is similar to that of Goldstein, except his incision is much farther back, and goes to the bone, thus making a thick sliding graft which he pulls over the fistulous opening.

Watson[11] makes a crescent shaped incision through the soft tissues down to the bone one and one-half inches behind the fistula,

elevates this flap, isolates the fistula behind and below, and finally cuts it free at the apex. A second flap is cut from the posterior meatal wall and stitched to the anterior margin of the fistula. The dissected fistula is now turned inside out and the raw surfaces approximated.

All of the above described operations are "bridging" operations. They do not attempt to even partially obliterate the cavity. They are applicable only to small fistulæ where scar tissue formation has been slight and where the surrounding tissue is healthy epidermis.

Straatsma[12] reports good results by use of a pedicle scalp flap, on the raw surface of which he plants a Thiersch graft. When this graft is firmly attached, the fistula edges are freshened and the flap turned downward into the fistula with the side of the graft toward the cavity and sutured on three sides, the pedicle side being left undisturbed for nourishment. After the flap has become attached to the edges of the fistula, that part of the flap not used to close the fistula is cut and returned to its original position on the scalp. The denuded area of skull is covered by a split graft from the thigh.

Frey[13] suggested the use of subcutaneous injections of paraffin to obliterate postoperative deformities, and to close postauricular fistulæ following simple mastoid operations. He used the method not only to fill the retro-auriclar depressions, but also to actually close any permanent openings.

During the World War, Eagleton[14] used bone grafts and bone chips for closing old mastoid wounds. He also was the first to mention the possibility of using fat for this purpose.

Straatsma and Peer[15] reported successful results following the use of free fat grafts taken from the thigh and abdomen, as suggested by Eagleton.

I have found the "tongue-flap" operation, which I devised, a very satisfactory method for closing postauricular fistulæ following both the simple and radical mastoid operations.

In this operation the entire scar and fistulous tract are excised and dissected out, leaving an eliptical cavity bordered on both sides by healthy epithelium.

A second incision is made parallel to, and one inch behind, the posterior margin of the wound, extending well above and below its upper and lower margins. This incision is through the skin, the superficial fascia, the extrinsic muscles of the ear, the thin lateral extensions of the Galea aponeurotica, the temporal fascia, the temporal muscle, fat and periosteum.[16]

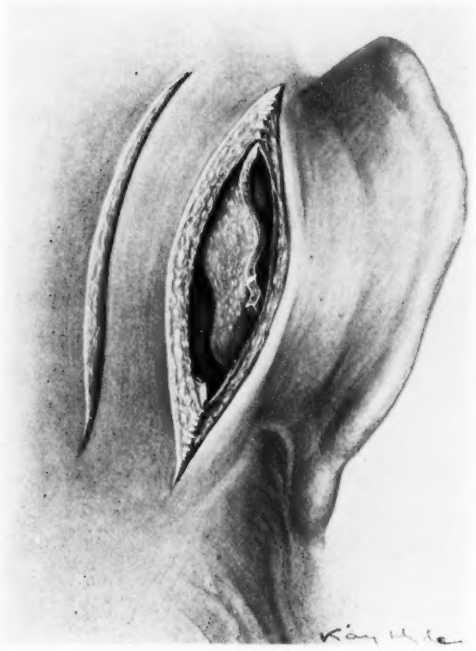

Fig. 4

The tissue between the two incisions is carefully dissected up from the bone throughout its entire length, making a flap one inch wide, attached at the upper and lower ends, but freely movable mesially and laterally.

The flap is then divided into two layers, leaving as much thickness as possible in the lower layer.

The lower flap is then cut free from the upper flap at its lower border, thus forming two flaps, the upper consisting mostly of skin and subcutaneous tissue attached at both ends, and the lower composed of muscle, fascia, fat and periosteum, attached only at its upper end, its lower end being entirely mobile.

The mastoid cavity is then freshened with a curet, and the muscle periosteum "tongue flap" is tucked into the cavity with the periosteum next to the freshened bone.

The skin flap is then moved over and sutured to the undermined anterior skin margin of the wound.

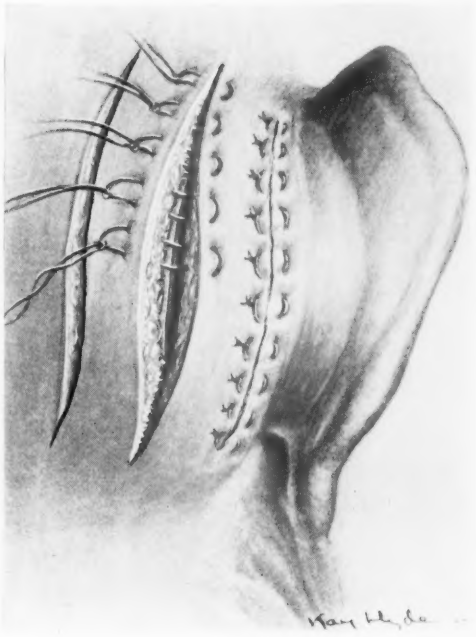

Fig. 5

To cover the denuded area of the skull, a third incision, parallel to and three-fourths of an inch behind the primary plastic incision, is made down to the periosteum; by undermining the edges of this third incision the wound's edges can be brought together and sutured without tension.

All wounds are closed with mattress sutures of dermal or catgut.

The routine mastoid dressing is applied and not disturbed for several days. Healing is usually by first intention.

The operation may possibly be contraindicated in individuals prone to form keloid scars and in those who, because of inoperable cells, have a continuous chronic discharge of pus through the fistulous opening.

The objection that an area of skull is stripped of its periosteum and may be subject to osteomyelitis cannot be sustained. Macewen's[17] experiments on regeneration of bone after removal of periosteum show that most of the osteoblastic tissue remains attached to the

bone even after the periosteum has been entirely removed. Clinically I have not seen any such symptoms develop.

The advantages of this method are:

1. The fistula is permanently closed.

2. The cosmetic results are excellent.

3. Tissue contiguous to the fistula is used in its repair, thus requiring only one operation.

4. The operation is simple and requires very little time to perform.

5. There is little danger of the wound breaking down, because of the rich blood supply and the well known rapid scalp healing.

6. The cavity is lined with periosteum, the natural covering of bone.

7. The flaps contain all the elements required for rapid healing and infection resistance—i. e., fat (important to healing, as pointed out by Hartwell[18]), muscle, good blood supply and periosteum rich in antibodies.

8. The operation is satisfactory for closing all types of post-auricular fistulæ, including those complicated by extensive loss of tissue.

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XLII

ENDOCRINE IMBALANCES AND THEIR RELATION TO THE UPPER RESPIRATORY TRACT*

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As scientific medicine has made its strides in the laboratory and as biologic chemistry has come to the fore, the individual practitioner at times feels more like a spectator than one of the team. Yet even to him there is no story more fascinating than that of the endocrines. The story has all the allurement of the mystery novel, without, however, the final chapter, for much of chaos still prevails.

It is unfortunate that commercial interests have invaded the therapeutic field, claiming specificity without proven merit, and reverting to shotgun formulæ which have no scientific foundation.

There is so much of the hypothetical written and discussed regarding the endocrine glands that it behooves one to first consider some of the things that we do and do not know about them. We know them as primitive structures having an essential part in Nature's scheme of life, birth, maturation, reproduction and eventual deterioration. As medical anthropologists we know that this system of glands with their incretions antedate the nervous system, and while acting more slowly, do maintain a certain reciprocal relationship. Yet their motor innervations do not seem to be of much clinical importance.

This system of glands is always in a state of dynamic balance or imbalance. Its incretions, known as hormones, as principles have specific functions. But as specific chemical compounds we know only a few. A multiplicity of hormones has been described as coming from some of the glands, yet, according to our present knowledge, the cytology of the glands themselves would be incapable of producing such a variety of specific substances. Furthermore, we do not know how hormones act. But we do know as a general principle that a hormone has no effect on the function of the gland that produces it.

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Ovarian hormones, for example, exert their influence not on the ovary, but primarily on the generative adnexa in addition to exercising a balancing influence on other endocrine glands. This is an endocrine axiom.

Imbalances are primarily associated with the hypo- or hyperfunction of one gland, but due to this interlocking system the clinical manifestation is often pluriglandular. This does not justify indiscriminate pluriglandular therapy but should serve as a stimulus to ferreting out the primary offender. The real therapeutic indication may be a hormone that stimulates or is antagonistic to the hormone-producing gland that is clinically conspicuous. Yet even this is not so simple, for the administration of a hormone, if one is available, may result in inhibiting its natural formation.

Perhaps it augurs well for the future of endocrine therapy that hormones do not seem to be species specific, yet animal experimentation is not conclusive proof that similar sequences will take place in man. We really know very little about the thymus gland. It has not been proven that it is a part of the endocrine system or that it produces a hormone. Endocrine symptomatology related to the thymus has practically very little clinical value, even though Timme gives a definite picture of the effects of subinvolution of the gland. Yet animal experimentation is quite striking. Tadpoles fed upon thymus gland suffer a marked delayed differentiation, and fowls deprived of their thymus gland will lay eggs without shells, and if later fed thymus gland will produce eggs with shells.

The physical and mental development of man is largely dependent on the harmonious balance between the endocrine glands. Being so essential to the scheme of life, the chronologic order of the manifestations of endocrine imbalance becomes of practical importance. Imbalances early in life may be overcome and obscure the clinical picture. Compensatory hyper- or hypo-function of other glands follows a primary disturbance and the resulting train of symptoms may entirely overshadow those directly due to the gland first involved.

The pineal gland, suggested as possessing antiestrogenic, antigonadotropic and growth inhibiting properties, has yet to be proven to have genuine endocrine function.

The pituitary, by some termed the master gland, is not entirely the master, for it also requires activation. It is, however, the great co-ordinator and activator.

As many as twelve hormones have been described as coming from the anterior pituitary, yet none have been isolated in a pure state. The gland has but two types of secreting cells: the basophilic and the eosinophilic. In spite of the cytologic limitations, five principles are generally recognized as coming from the anterior pituitary, namely, the somatotropic, gonadotropic, thyrotropic, lactogenic and interrennotropic.

Pituitary dysfunction rarely manifests itself with respect to a single hormone. Even more general acceptance is being accorded the hypothesis that the pituitary directly or indirectly affects fat and carbohydrate metabolism and that there is an endocrine antagonism to the islands of Langerhans. Principles from the anterior pituitary have an accelerative effect on the thyroid, the gonad and the suprarenal cortex, while they in turn have a depressant effect upon the pituitary function. The gonadal principles from the pituitary consists of two factors, one follicle stimulating and the other luteinizing in effect. The clinical application of hormones from the anterior pituitary is at present confined to the growth and gonadotropic factors. The so-called anterior pituitary like hormones obtained from pregnant urine cannot be said to be identical with the pituitary factor, in that they do not produce complete follicle maturation and in the male effect only the interstitial cells. The principles obtained from the urine of the castrate and after menopause conform more nearly to the true pituitary principle, lacking, however, the luteinizing factor. It is possible that if and when there are available gonadotrophic hormones from the anterior pituitary the clinical results will be much more satisfactory.

From the posterior pituitary two fractions possess striking clinical properties. Cytologically the posterior lobe consists of pituicytes, neurogliar cells, and nerve fibers with the first only regarded as secreting structure. The one factor or hormone, pitressin, has pressor powers; the second, pitocin, causes striking oxytocic activity, and a third has antidiuretic properties. This power of pitocin is held in abeyance during the early stages of pregnancy, probably by a hormone from the corpus luteum, which acts as an inhibitor.

The suprarenal gland is essentially an anatomic union of two glands, the medulla, originating from the neural ectoderm, and the cortex, from mesoblastic tissue. No established functional relationship exists between the two. No specific function has been proven for the incretion from the medulla, though its predominate action is on tissues that receive active innervation from the sympathetic nervous system, serving as an activator and synergist to this part of

the autonomic system. It is in no way essential to life, and the prominent position that it has gained through its potency as a vasoconstrictor and retarder of capillary circulation as applied in asthma, serum sickness, angioneurotic edema and urticaria, is due to its pharmacologic action and not as an endocrine replacement.

The cortex is essential to life, but in just what way we do not definitely know. Its hoped for therapeutic success in Addison's disease has not been fully realized, though it is of undoubted benefit in acute crises. Its hormone, cortin, has been isolated in crystalline form by Kendall, but his product has not been generally accepted as a single chemical unit. The function of the cortex is still veiled in mystery. There seems to be a relationship to the productive and integumentary systems, even constituting a special pituitary adrenal gonadal interrelationship. Normal bodies seem in no way affected by the injection of cortical extract. Hyperpituitaric states lead to adrenal hypertrophy, in which process the thyroid and not the thyrotrophic principle from the pituitary is essential. Sodium excretion, carbohydrate metabolism, muscular weakness and asthenia have been studied in their association with the cortex. Certainly the fact that cortical extracts have been able to maintain adrenalectomized animals established its position as one of prime importance, yet the indiscriminate administering of cortical extracts does not as yet rest on a scientific basis.

If we designate the pituitary as the master co-ordinator, certainly the thyroid demands recognition as the predominating endocrine gland in direct action on body tissues. It is the master catalyzer. Yet hyperplasia, denoting hyperactivity, does not necessarily mean hyperfunction. There is no definite proof that its association with, also means being under control of the sympathetic nervous system. As far as is known, it is the only organ known to manufacture an iodin containing hormone thyroglobulin. Yet we know very little of the formation of organic combinations of iodin or how the effects of its oxidative influence are accomplished. The benefit of Lugol's solution in hyperthyroid states is unexplainable. Its nerve connections are not essential, for thyroid transplants will receive equal stimulation. It is directly activated by the pituitary, and without this stimulus it atrophies. Conversely, its hormone depresses the pituitary. Exophthalmos is no longer considered primarily the result of thyroid hypersecretion. A somewhat similar relationship exists between the gonads. In fact, the relationship is triglandular, between the pituitary, the thyroid and the sex glands, with the suprarenal cortex as a probable additional factor. An antagonism exists between

the thyroid and the pancreas. Pituitary response may be similar in exophthalmic and endemic goiter, but not for the same reason. In myxedema and cretinism, thyroid feeding is purely a substitution therapy.

The parathyroids are concerned with calcium-phosphorus metabolism and with the preservation of a normal nervous system and normal muscle contractility. Its active principle raises blood calcium and increases the excretion of phosphorus. Whether vitamin D serves as a complete substitute for the parathyroid hormone can not be stated, because the presence of accessory parathyroid glands prevent completely positive experimental extirpation of parathyroid tissue. Determination of the level of blood calcium is the one means of regulating hormone dosage. Overdosage means possible kidney damage and kidney stones, accumulation of nitrogenous waste products and a depletion of calcium from the osseous system. The parathyroid hormone has a rapid but transitory effect which limits its use to rather acute conditions. With vitamin D a similar effect is produced gradually and with the disadvantage that in the case of overdosage the hypercalcemia continues for two weeks after discontinuing the vitamin D.

The product of the pancreas, insulin, enables more sugar to be burned, resulting in increased oxygen consumption and a higher respiratory quotient. But how this is done we do not know. Does diabetes result from an overproduction of sugar or a lack of ability to use sugar? This is not known. Do these products of the cells of pancreas, which apparently do not depend on nervous control, result because of increased sugar content of the tissues or do they themselves regulate the discharge of dextrose from the liver? We do not know. We do know that the thyroid, the pituitary and the adrenal cortex have an antagonistic action relative to the pancreas.

The three most important endocrine deficiencies, those of the thyroid, the pituitary and the gonads, have accompanying nutritional disorders that are unexplainable. They may be divided into adipose and nonadipose groups. Thyroidectomy may be followed by a cachexia or pseudo-obesity and thyroid extract will relieve either.

Heredity has a definite responsibility in relation to the endocrinopathies. Endocrine balance is also affected by prolonged environment, therefore the training of the infant as to correct habits and surrounding him with a regular environment has a practical significance.

The chronologic order in which symptoms have appeared are of great importance, and the period divisions, as stressed by Engelbach (infancy, 1-5; juvenility, 6-13 or 14; adolescence, 13-20, and maturity), if followed, will aid greatly in clarifying the clinical picture.

Endocrinopathies of the infantile and juvenile period are commonly of the hypofunctioning type. The reverse occurs during adolescence. During infancy the thyroid, the parathyroid and the thymus glands rank in the order named as to frequency of disturbed function. The thyroid assumes a position of major importance, because it is concerned with the embryonic differentiation and the postnatal development of the endocrine system. Early recognition lessens the chances of progression. The infantile hypothyroid is often overweight at birth, and this is followed by a delay in dentition and the ability to walk and talk. Retarded mental development at an early age more often is the reason for bringing the patient under medical observation. A reduced metabolic rate and its determination is important to all thyroid groups, but of less practical importance in infancy because of the difficulty of its determination. The delayed development of osseous centers in the knee and ankle form most important diagnostic aids. This delay, together with maldevelopment of the genital tract, differentiate the condition from mongolism. The obesity in the infant hypothyroid is recessive in type. Hypothyroid conditions in juvenility have generally been projected over from infancy. A myxedema from exogenous causes is an exception. The juvenile and adult types participate in the common classic facies, the alabaster color, mucin infiltration of tissues, dermal, hair and nail changes. Dorsal padding, which is quite indicative, is unexplainable. The hypoactivities of the thyroid and also of the pituitary are characterized by a rough, dry, scaly skin, while the opposite epidermal condition accompanies their glandular hyperactivities. After juvenility the hypothyroid condition tends to become biglandular in type. Because of the antagonism between the thyroid and the gonads, we may expect an early appearance of menses and in the adult a delayed climacteric. During adult life the hyperactivities of the thyroid assume an important position, producing the well known symptoms. The pituitary function during infancy and juvenility is largely directed towards growth. Endocrine dysfunction during these periods is principally confined to the thyroid and the pituitary. The importance of the growth factor is self evident, since the body gains one-half its total height by the end of the third or fourth year

Adiposity is usually the predominant and the earliest sign of pituitarism, is not recessive, and manifests itself in late infancy and juvenility. Here we do not have the retarded mental and physical

development as in the congenital hypothyroid. The clinical picture may, however, be one of over- or undergrowth, with or without adiposity. The juvenile hypopituitary disturbances are largely unrecognized until the age of gonadal activity.

The adiposogenital pituitarism is alone in causing a concomitant obesity and a genital aplasia. The obesity is characterized by a girdle, mammary and mons obesity. During adolescence the symptoms are identical but with a more pronounced underdevelopment and a deficient function of the genital system. During juvenility and projected into adolescence, pituitary dysfunction also tends to become biglandular in type.

Puberty demands a complete endocrine readjustment. The termination of adolescence is determined by the final epiphyseal unions. The pituitary is not alone in possessing a hormone growth factor. The gonads primarily influence the fusion of long bone epiphyses. Likewise the suprarenal cortex has functions affecting growth, genital function and metabolism. Pituitary tumors have been greatly overestimated as a cause of pituitary disturbance and they seldom occur before the thirtieth year.

Statural proportions assume an important diagnostic value during adolescence. The different types of giantism, normal eunuchoid and acromegalic, are to be explained by the relative amount of overproduction of the growth hormones, the sex hormones and the age period. The distinction between gonadotropic and estrogenic hormones is at times confused. The gonadotropic effect of the pituitary indirectly brings about estrogenic effects through stimulating the gonads to produce estrogenic substances. Estrogenic substances are essentially growth principles affecting the genital adnexa and the mammary glands. The presence of estrogenic substances in large amount in pregnancy is little understood. Gonadotropic hormones also appear in large amount and with greater rapidity than even estrogenic substances. They are present in a few days after the first missed period and account for the successful practical application of the Ascheim-Zondek pregnancy test. After parturition the excess of gonadotropic hormones disappears in three days.

An unusual gain in height during adolescence with an increase of lower measurements over upper measurements and accompanied by a hypoplasia of the genital tract is characteristic of primary hypogonadism. Primary hypogonadism has no proven definite relationship to the hypophysis. This statural disproportion results because final epiphyseal union is under the direct influence of the gonads. A state

of hypergonadism before adolescence is usually due to an over-active suprarenal cortex. In contrast hypergonadism during adolescence is characterized by early epiphyseal unions with consequent disproportionate increase of upper measurements over lower. As a result, the individual has a stocky build with an excessive muscular development, at times mistaken for obesity. The two sexes vary in general symptomatology, the female showing a tendency to an inversion to the male type and a hypertrichosis. Such tendencies are not prominent in the male, virility being the outstanding characteristic.

The comparison of height to span and upper to lower measurement differentiates pituitary from gonadal disturbances. Anatomic abnormalities are of much less importance in the adult than in the adolescent period.

The selective location of hair in the two sexes is unexplainable in glandular disorders. In the early castrate and eunuchoid there is a hypertrichosis. Localized baldness of scalp and eyebrows often accompanies hypothyroidism. Hirsuitism, due to the suprarenal cortex, is generalized, involving the body and face. This facial hirsuitism is especially prominent in the female. An overgrowth of hair on the distal extremities, associated with vertex alopecia, is significant in acromegaly. In the hypogonad the lack of hair affects the face but not the scalp.

Equally unexplainable are the obesities accompanying endocrine imbalances. There is supraclavicular and dorsal padding in the hypothyroid. The girdle adiposity in the hypopituitaric and the trochanteric padding in the hypogonad are particularly striking. Again there is no explanation for the fact that there is an absence of adiposity in the castrate until after thirty.

Dermal changing, such as the bronzing in Addison's disease, chloasma and vitiligo in pituitarism, pigmented warts in acromegaly, and the erythemas in adiposo-genital pituitarism are cited as clinical facts without satisfactory explanation.

In hypogonadal conditions, particularly in the adult, severe gastro-intestinal symptoms will at times obscure signs of the endocrine imbalance as the real cause.

The mucous membranes of the upper respiratory tract in its exposed position reflects many exogenous influences but perhaps of equal importance are its reflections of various metabolic disturbances. These are not easy to interpret.

There has been a tendency to inspect these membranes and then draw conclusions as to certain metabolic disturbances as definite causative factors. There is much to commend such investigations, but unusual care must be taken to avoid drawing erroneous conclusions.

The purpose of this paper was to approach the mucous changes in the presence of known metabolic disturbances, due to endocrine imbalances. This is difficult in private practice where the opportunity to contact such patients is limited.

Wm. R. Hunt of Santa Barbara made a tabulated correlation of ear, nose and throat reactions in 659 cases of endocrine disorders; with the exception of those conditions directly referable to osseous development and architectural changes there were few conditions that could be directly referred to a primary endocrinopathy as a cause. An excess of lymphatic tissue was noted in 68 patients. In 34 of the pituitary group and in 17 of the gonad group this occurred on the posterior part of the tongue.

Reviewing 96 cases of Engelbach, his patients were subjected to a careful rhinologic examination, the color of the mucous membrane being mentioned quite frequently, yet the positive findings as affecting the upper respiratory passages are remarkable in their absence as far as being attributable to an endocrine imbalance.

In the group studied for this presentation, made possible by the Department of Otolaryngology of the College of Medicine of the State University of Iowa, 81 patients were studied, 39 of the hyperthyroid, 6 myxedema, 7 of the hypothyroid, and 21 of the diabetic group. The others varied and were too small in each group to be of study value.

As to the color of the nasal mucous membrane the hyperthyroid group tended to some increased redness of the same, but only in about 50 per cent of the patients, and this was not of sufficient intensity to be of marked significance. The hypothyroid group tended more markedly toward the pale side, 80 per cent. Rather misleading, however, was the fact that in the six cases of myxedema the nasal membrane was toward the red side in three and toward the pale side in three. Among the diabetics (21) thirteen were towards the red side and five toward the pale side. There were no conclusions to be drawn regarding the degree of moisture, mucopurulent bridges, dried secretions and sensitivity of membranes except there was an increase of moisture in all the hypothyroid group (7). No allergic history was revealed in the hyperthyroid (thirty-nine) or the diabetic (twenty-one) groups, but three out of the seven of the hypothyroid group

gave a positive allergic history. Seventy per cent of the hypothyroid group showed a paleness and bogginess of turbinate tissue and a marked postnasal discharge in six out of seven. An ethmoiditis was present in six out of the seven hypothyroids, but in the other groups not often enough to be of significance. The postpharyngeal wall, the lingual tonsil and the larynx revealed nothing significant.

In the hyperthyroid group the appearance of the pharyngeal tonsils and the presence of chronic infection, at times accompanied by a redness of the anterior pillars, was rather striking. Among the thirty-nine, and this was an adult group, the tonsils had been removed in ten because of infection, and twenty-four showed marked infection, this giving a ratio of 81 per cent. Eliminating the nodular goiter and the exophthalmos without goiter groups, the ratio rose to 90 per cent. In the hypothyroid group the ratio was 57 per cent, and the diabetic group 61 per cent.

Conclusions are principally of a negative nature. A paleness of the nasal mucous membranes with boggy turbinates associated with mild lethargy should suggest a possible hypothyroid condition. Vasomotor rhinitis and allergy may simulate the same picture.

Chronic pharyngeal tonsillar infection has a close association with hyperthyroid conditions, particularly where toxicosis is prominent. Whether it preceded or is a sequela I am unable to state. Excepting the above, the endocrinopathies seem to offer little as causative factors in abnormal states of the upper respiratory tract. The thyroid, because it is the one endocrine gland whose hormone has a direct oxidative effect on general body tissues, may be the possible exception.

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XLIII

ADENOIDS AND IMMUNITY*

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In my previous papers[1 2 3] I took up the morphology and structure of the adenoid, drawing attention to its symmetrical form and the effect of its regularly disposed lobes and valleys to give as large a surface area in the pharynx as possible, the purpose of this large exposed area probably being to absorb and attack part of the infected secretions, which drop back from the nose. I drew attention to the fact that there are no pockets or foci of pus or infection in the adenoid as in the case of the tonsil. I gave my clinical experience in over 500 cases in leaving the adenoid undisturbed when tonsillectomies were done and in treating the nasal obstruction and infection by means of washes and blowing exercises, and noted that the return of these patients for ear conditions was less frequent than those cases which had tonsillectomies performed and had not used nasal hygiene.

I have been encouraged by the reports of those who have carried out this plan of conservation of the adenoid and who have spoken or written to me. Much skepticism generally remains, however, as to the usefulness and as to the harmlessness of the adenoid.

The strategic position of the adenoid in regard to upper respiratory infections deserves consideration. The general toxemia resulting from acute infections of the nose is often greater and sometimes less than the nasal condition would seem to warrant. Perhaps an indication of how infection is absorbed is obtained from an experience familiar to all of us.

In the use of cocain in preparing for a nasal operation I have observed that the symptoms of the absorption of the drug becomes evident only as the cocain reaches the nasopharynx. So long as the drug is confined to the nasal mucosa, cocain mud and packs of strong cocain solutions are usually well borne. If, during the applications, the posture of the head is not inclined forward and the cocain drops back to the nasopharynx, the stimulation of the sympathetic at once becomes evident.

This common experience points to a possible interpretation of the effects of an acute nose and throat infection. Gravity and the

*Read before the Western Section meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Diego, Cal., January 30 and 31, 1937.

conformation of the nasal cavities make for the flow of nasal secretion to the pharynx even in the erect position. The infection starting in the nose drifts back to the pharynx, and here absorption of the bacterial toxins takes place, producing systemic reactions either of resistance, (higher temperature and pulse rate) or of depression.

It is true, symptomatically, that the sore throat is often complained of before there is much reaction in the nose, but this sequence is, I think, only apparent. A latent infection of the nose may become acute, and in the early stages of this acute phase cause no symptoms in the nose itself. As soon as the activated bacteria in the nasal secretions drift backward and reach the nasopharynx and pharynx, absorption and general reaction take place. With such a sequence continuing for countless generations in many animals, defense should develop at the point of entrance of the infection. The defense against infection of a mucous surface is surface lymphoid structures. In the pharynx are adenoids, tonsils or patches and pillars of lymphoid material, in the intestinal tract are Peyers patches and the innumerable solitary lymph follicles. The placing of these structures, their morphology, their structure and their reaction to bacterial infection is so suggestive that the conclusion that their function is the combat of surface infections by the production of antitoxins, precipitins and other antibodies seems unavoidable.

Perhaps a broader view of the structure and function of lymphatic organs can be obtained from the literature of general pathology and general medicine than from textbooks and articles limited to the discusison of the nose and throat. We have so long considered the lymphatics of the throat as pathologic, and the subject for immediate surgery that it is well to stand away from our specialty and consider the essential nature of lymphatic structures. Since the first recognition of adenoids by Wilhelm Meyer* there has been appar-

*I was fortunate in finding Wilhelm Meyers first English paper[1] reported in the Lancet, 1869. In all the literature I have read I have not found one correct reference to Meyer's original work, two of the references even giving his first name wrong. I will quote his description.

"These vegetations, when examined microscopically, are found to be composed of the so called adenoid tissue and are accordingly to be regarded as overgrowths or morbid growths of the closed glandular structures allied to the lymphatic glands found naturally in or beneath the mucus membranes of the pharynx, the fauces and the base of the tongue."

The paper was presented without Dr. Meyer being present and aroused considerable speculation. Mr. Smith and Mr. Heath wondered if it might be the same as the pharyngeal tonsil which they had been able to observe very clearly in cases of cleft palate. Mr. Marshall had seen much hypertrophy but nothing he considered a morbid growth.

ently no question raised as to the extent their mechanical obstruction is deleterious, nor more recently has it been seriously questioned whether they were foci of infection. Inclusions of pus are found only rarely. They take the form of quiescent, apparently quite inactive cold abscesses.

In my literary research I found no work on the effect of infection on surface lymphatics, but the following quotations in regard to lymphatic nodes must be true with but slight modification in regard to surface lymphatics.

Drinker, Field and Ward,[5] after experimental infection in dogs, conclude: "The nodes are held to provide two sorts of filtration, the first of simple mechanistic type, and the second biologic, due particularly to the phagocytic activity of the reticulo-endothelial cells."

These authors also say in regard to the filtering efficiency that this is "So great as to make it fairly certain that in a part kept at rest early in an infection practically no micro-organisms would escape the nodes in the line of drainage." Boyd[6] states in regard to the reticulo-epithelial cells that they are "distinct from the endothelial cells of vessels (and) have great phagocytic properties. Some of these (however) line vessels especially in hemopoetic organs and others are supporting or reticular cells. Their function is (1) phagocytosis, (2) storage, (3) antibody formation, (4) formation of blood cells. Their action is twofold. In the first place they are the most actively phagocytic cells of the body. In the second, they appear undoubtedly to play a part in the production of antitoxins, precipitins and other antibodies. It is in the chronic form of infection that these cells are most in evidence."

Common experience shows that his remarks in regard to nodes may be applied to surface lymphatics. "Chronic lymph node hyperplasia will occur whenever the lymph nodes are in the path of absorption of some chronic irritant. This may take the form of bacteria of low virulence, toxins or foreign particles."

He remarks the importance of these organs, "For the lymph node is not a mere collection of lymphoid cells. It, like the spleen, is a reticulo-endothelial structure containing lymphoid tissue." And in speaking of germinal centers he says, "It is probable, therefore, that these structures, germinal centers, are composed not of lymphoblasts but of modified reticular cells.

Bailey's Histology states:[7] "The injection of nontoxic dyes, such as trypan blue or lithium carmine, into a living animal is known as vital or intravital staining." Those cells taking this vital stain are

known as the reticulo-endothelial cells, and their function in taking care of bacteria and toxins is indicated by their action in taking up the foreign material of the dyes.

Sherwood[8] says: "Renewed interest in cellular immunity was stimulated by the work of Aschoff on the reticulo-endothelial structure (1924) and of Besredka (1927) on local immunity. According to Maximoro (1924), the reticulo-endothelial structure is a vast system of cells throughout the whole body representing the mesenchyme carried over into the adult and remaining undifferentiated throughout life. Hence it retains its embryonic potentialities of differential into many different forms under different conditions."[9]

Kolle and Hetch:[10] "A combination between the reticulo-endothelium and the formation of antibodies is proved by facts which have long been known, but it would be quite unjustifiable to look for the dominant or sole causes of active immunity and of antibody production in this system," but a few pages later[11]—"The notable amounts of toxins stored in the spleen indicate the importance of the reticulo-endothelial structure in the processes of immunization."

Surface lymphatics differ from lymphatic nodes mainly in morphology and in the manner in which infected material reaches them. The morphology and significance in the adenoid I have already stressed.[1,2,3] In studying the physiology we consider first its enlargement. That the adenoid increases in size during acute and subacute nasal infections is common knowledge. This is only a temporary increase and subsides markedly after the conditions causing it subside or are relieved, exactly in the way lymph nodes return to normal size after the distal infection subsides or is relieved. This enlargement of the adenoid is functional and is a physiologic reaction to an infection usually starting in the nose and sinuses. Infection is carried to the lymph nodes by means of afferent lymph and blood vessels. In the laboratory it is easy to send vital dyes in the same direction and through the routes that infection reaches the node in life. The effects of an infection and the infection itself do not reach the surface lymphatics by means of afferent vessels but by direct contact with the surface of the gland.

The literature revealed no experiment on vital staining in the study of physiologic activity of surface lymphatic glands. In the time at my disposal I investigated the different dyes in regard to their toxicity, penetration, etc. I found the vital staining of the nodes of little value in staining surface lymphatics. The dyes used are too attenuated and the foreign material (India ink) does not readily pen-

etrate the mucous membranes that cover the surface lymphatics. Fenton (?) found in his recent experiments that weak stains produced no result on the glands of the alimentary tract. Much more concentrated dyes are necessary and, in addition, penetration must be aided by reducing the surface tension. After correspondence with dye manufacturers and laboratory workers, consuming a great deal of time and being absolutely inconclusive, I determined on the commercial preparation of bismuth violet.

I tried dropping this solution through the nose of several children, lying in a dorsal recumbent position for about an hour previous to their operation for removal of tonsils and adenoids. Both the tonsils and adenoids stained well superficially, but not deeply enough for any useful information to be obtained from study of the sections. If the staining were carried out daily, perhaps for a week previous to operation, it is possible the stain would be carried deeper and the cell reaction could be studied. My attempts in this direction were not successful on account of the objection of the parents and their failure to follow directions. I decided that, in order to accomplish this effectively, it would have to be done in an institution or in a hospital where the child about to be operated on would be under competent treatment and experimentation.

Animal experimentation, bacterial toxins or India ink could be incorporated with the dyes and the effects noted. However, this is a virgin field and the technic would have to be worked out by trial. Though I spent considerable time and care on my own experiments, they were inconclusive, and this work will necessarily have to be carried out where clinical and laboratory facilities are available.

In conclusion, the morphology, position, the postoperative findings of the adenoid and the clinical experience of many observers strongly suggest that the adenoid plays some part in the production of immunity in upper respiratory infections. The newer general pathology and physiology of glandular structures also supports this view.

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XLIV

ETIOLOGY OF THE SADDLE NOSE: PRELIMINARY REPORT*

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PHILADELPHIA

A comprehensive review of the literature dealing with the etiology of the saddle nose is rather surprising. Some eminent writers inform us that this deformity is due chiefly to syphilis[1] while equally brilliant authors state that trauma[2] is the principal cause. In both instances other factors are mentioned, such as infections, faulty intranasal surgery, local manifestations of syphilis,[3] tuberculosis[4] or malignancy[5] and a host of lesser and rare causes. The deformities resultant therefrom vary according to the pathology, the amount of tissue destruction and the amount of tissue contraction. The tertiary lesions appear from three to twenty years after the primary manifestation—and are very destructive to both soft and bony tissues.[6] Perforations of the septum due to gummas[7] are posterior—those anterior are either traumatic, postoperative or tubercular.

Prior to the World War, cosmetic plastic surgery was in its infancy as far as the general public was concerned. I mention this advisedly, for after all, the public creates and demands the specialist. Very few in this country practiced this specialty exclusively, and so very few nasal deformities were corrected from a cosmetic viewpoint. During this pre-World War period some of these patients gave a history of syphilis or it was detected by the laboratory. Even those with a negative Wassermann were considered luetic. Most of these individuals also gave a history of trauma which seemed to be of little importance, except that it excited the onset of the destructive process. Syphilis is capable of producing saddle deformities of various degrees, from the very mild to the most destructive types. This accounted for the reasoning that most saddle noses were directly traceable to syphilis.

Up to this writing a careful search of the literature has failed to unearth any definite statistical data[8] or to swing the etiologic factor or factors in another direction.

*From the Department of Plastic Surgery, Eastern State Penitentiary, Philadelphia, Pa.

From the records of the Eastern State Penitentiary, where I have had the opportunity of correcting many of these saddle deformities, the statistical data collected after reviewing the records of over 3,000 inmates forms the bulk of evidence which stimulated the writing of this article. At all times more than 200 inmates are being treated for syphilis. An additional 100, having received sufficient treatment, are Wassermann negative. The latter cases have a Wassermann test at regular intervals to insure a serologic cure. If the blood becomes positive at any time the patient returns to the former group when active treatment is again instituted.

After a most careful search we† found six cases with either active or latent syphilis with nasal deformities closely resembling a saddle nose. Further examination revealed only one inmate with a distinct saddle nose directly traceable to syphilis. This individual had a persistently four plus Wassermann which was present at the time of his admission to the institution. He had had considerable treatment with little or no change in the serologic picture. Intranasal examination revealed a total destruction of the cartilaginous together with most of the bony septum. The nasal bones themselves did not appear to be invaded and seemed to be clinically negative. Despite the total erosion of the cartilaginous septum, the external deformity was not marked. Is this a case of malignant syphilis?[9] Did the history of trauma (several street brawls) lessen the resistance of these structures to the ravages of the spirocheta pallida? Is the amount of trauma required in producing a saddle deformity the same in both the luetic and the nonluetic patient? This and many other questions are still being weighed in the balance. We do know, however, that the type of saddle nose with a developmental dystrophy will sustain operative trauma far better than one which has been attacked by the spirocheta pallida. It might be interesting to record we found a distinct saddle deformity, which disclosed an anterior septal perforation with complete destruction of the columella, in a cocain snuff victim. From the strata of individuals housed in our penal institutions, one might expect the incidence of syphilis and saddle deformities resultant therefrom to be quite high. From the foregoing it appears that syphilis as a direct causative agent is contradictory to our findings. This extremely low incidence of syphilis productive of a saddle deformity among so many luetics in a penal institution is indeed interesting. It is true, we encountered many saddle noses but they all were Wassermann free and all gave a history of trauma ranging from accidental injuries to blackjacks. Some congenital types were found,

†I wish to express my indebtedness to the Resident Physician, Dr. Frank J. Jodzis, for his assistance in collecting this data.

while many stated that they possessed a "good looking nose" until after a submucous resection, when the nose later "caved in."

In private practice I have encountered only one case of a saddle deformity with a positive Wassermann; all others were negative. Etiologically the deformity was either congenital or due to the various types of trauma, with or without infection. Trauma due to external violence, traumatized septal cartilage resulting in a saddle deformity or a septal abscess following a submucous resection is the most common history elicited from patients. In the above singular instance the patient was a college student (Wassermann plus four), whose nose became distinctly concave after several nasal injuries incident to his athletics. After consulting his physician (a most reputable urologist) he assured me that the patient had received adequate treatment despite the persistent Wassermann and that the operation might be performed without any great danger. With this assurance, a two-stage saddle correction[10] was accomplished with an excellent cosmetic result. With the average patient, in the event of a positive Wassermann reaction, it is the better part of wisdom to withhold operation until a negative phase has been reached. If the blood remains persistently positive, despite adequate treatment, the operation may be attempted, as in the above instance, swinging the pendulum of responsibility on the patient.

Some years ago I saw a patient with complete destruction of the bony and cartilaginous septum without any appreciable saddling. Effler[11] of Toledo has had a similar experience and no doubt others have viewed a similar picture. Without claiming originality, although no statistical data could be found in the literature, I am convinced that trauma plays the stellar role in the production of the saddle nose and not syphilis. Others with whom I have either spoken directly or been in communication are in accord with this view; among them are Hardy of London, Kazanjian of Boston, Straith of Detroit, Safian and Maliniak of New York and Cohen of Baltimore. Present day methods of anti-syphilitic treatment have made such deformities comparatively rare.[12] The hereditary influence in determining the size and shape of the nose is a well established fact. This Mendelian characteristic may be present in most members of the same family or present some atavistic tendency. A slight concavity of the nose, commonly referred to as the retroussé type, is, in most instances, a familial or racial characterstic[13] and is not to be confused with saddle deformities. Undue prominence of the nasal tip may resemble a saddle nose. These suggestive types must not be classified as luetic without definite confirmatory evidence. The upward and outward

flare of the nostrils incident to a depressed nasal bridge, so frequently seen in hereditary syphilis, is just as frequently encountered in nonluetics. The vast majority of these deformities are directly due to violence of some sort or infection following a submucous resection. A collapse of the nasal bridge with a perforation of the septum and a flaring of the nostrils is such a frequent sight that one may venture the question regarding some intranasal procedure without treading on thin ice. Moreover, it is highly probable that a slight displacement of the nasal bones before ossification is complete may continue to grow out of alignment or its growth may be retarded and, not reaching its full maturity along with the rest of the structures of the nose, may result in a developmental dystrophy, a saddle nose.

SUMMARY

1. Syphilis is a contributor to the causation of a saddle deformity, but it rarely occurs without trauma, except in the congenital types.

2. A casual glance into a luetic dispensary will reveal saddle deformities as an exceedingly rare sight.

3. The incidence of syphilis among patients with all types of saddle deformities is very low.

4. Infections, the granulomatas and other diseases, by their destructive processes, are capable of producing a saddle nose, but the number of such cases is small.

5. Trauma is by far the most important etiologic factor capable of producing a saddle nose.

WIDENER BUILDING.

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XLV

A GENERAL CONSIDERATION OF DEFECTIVE HEARING AND DEAFNESS WITH PARTICULAR REFERENCE TO ETIOLOGY. PART II*

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The physiologic differences in acoustic pressure of labyrinthine fluid between the windows play on the fibers of the membrana basilaris and through them stimulate the hair cells in Corti's organ and result in sound perception. According to Meyer,[1] the periodic difference in pressure between the windows is greatest at the moment when external air density changes most rapidly. Normal hearing, therefore, is the result of acoustic oscillations of the stapedial footplate produced by sound waves from the surrounding atmosphere directed against the membrana tympani and conducted to the oval window through the tympanic ossicular chain and the cranial bones. In the first part of the auditory excursion the footplate of the stapes is displaced inward and the pressure of the labyrinthine fluid is increased and displaced toward the round window at the base of the scala tympani with consequent increase of pressure in that locality and outward bulging of the round window membrane. The second part of the auditory excursion is a reversal of this movement and a decrease in labyrinthine pressure. For protection to the fragile labyrinthine structures against severe and what otherwise might be dangerous intensities, and for maintaining a constant physiologic tensive equilibrium, nature has provided an expansion chamber, the aqueductus cochleæ, which acts as a safety valve by permitting escape of perilymph into the subarachnoid space at a point near the jugular foramen where the perilymphatic and the cerebrospinal fluids mingle.

There has been a general concurrence in the assumption that labyrinthine pressure is affected only by acoustic changes of intratympanic pressure. This is because of the generally accepted belief that the labyrinth is well protected by its osseous capsule against nonacoustic pressure changes, and it has been assumed, therefore, that the labyrinthine fluid is endowed with a very nearly constant hydro-

*Read before the annual meeting of the Tennessee Academy of Opthalmology and Otolaryngology, Knoxville, April 12, 1937.

static pressure, the only physiologic variation, which is slight, being that provided for by the aqueductus cochleæ.

Kobrak[2] doubted the feasibility of the assumption that only intratympanic changes in pressure resulting from acoustic vibration could be transferred to the labyrinth. Therefore, he undertook to determine experimentally whether or not this assumption is the correct view. He conducted extensive experiments on dogs and rabbits, including both dead and living animals, by a very complicated and delicate technic. He clearly demonstrated that a change of 2 cm. of water in intratympanic pressure displaced 0.02 cmm. of labyrinthine fluid. He observed also that a very much greater rise in intracranial pressure was required to produce an equivalent change in intralabyrinthine pressure. He extended his experiments further to determine the effect on the hearing produced by changes in tympanolabyrinthine pressure. To accomplish this he exposed the tensor tympani muscle and subjected it to an audiometric tone of only sufficient intensity and of a definite pitch that would cause the slightest discernible muscular contraction—that is, threshold contraction. He noted that slight changes in middle ear pressure did not cause abnormal responses in the contractions of the tensor tympani, whereas any considerable abnormal increase in degree of either positive or negative intratympanic pressure caused very definite impairment of functional muscular response, and this produced abnormal pressure changes in the labyrinth. The phenomena observed in this interesting research appear to justify the conclusion that the hearing may be impaired proportionately to the sustained and long continued pathologic increase of either positive or negative intratympanic pressure.

The slight rise in intralabyrinthine pressure, as compared with the much greater rise of intratympanic pressure, may be accounted for by the presence of the aqueductus cochleæ. While extremely slow changes in pressure may be transferred entirely through the aqueduct, it seems that higher frequencies of pressure changes do not pass freely through this canal. Apparently it is upon this phenomenon that the function of normal hearing depends, because if all changes of every degree of frequency in fluid pressure were transferred freely and instantaneously through the aqueduct there could be no functional stimulation transmitted to the membrana basilaris and, consequently, there could be no hearing. The fibers of the basilar membrane are adjusted for length, tension and load, like the strings of a musical instrument, so as to function in response to sound stimulation throughout the range of a musical scale comprising about eleven octaves, which is the normal range of human hearing. At the apex of the cochlea the fibers of the basilar membrane are about three times

as long as they are at the base, and this difference in length is computed by some competent observers to be sufficient to account for about two octaves. The graduated difference in tension, which increases as the fibers decrease in length, accounts for about seven octaves, and this is the most important factor in nature's provision for the wide range of normal hearing. Finally, loading the fibers by fluid contained in the scala tympani and, as many observers believe, mass vibration of sectors, particularly sectors of the longer fibers, add two octaves in the low tones. This complicated arrangement accounts for the total wide range of eleven octaves of normal hearing. The fibers of the basilar membrane analyze sound into its component harmonic parts which are converted into nerve impulses and transmitted through the hair cells of the organ of Corti and the acoustic nerve to the auditory center in the brain cortex where all sound is interpreted.

Therefore, any pathologic alteration that primarily affects intratympanic pressure, either negatively or positively, and secondarily produces corresponding changes in intralabyrinthine pressure; or any pathologic alteration that produces fluid pressure changes in the labyrinth primarily, if sufficiently advanced and sustained, may affect the membrana basilaris by producing changes in, or more or less complete abolishment of, the tone response of its fibers. It follows, then, as a pathologic sequence, that in the course of progressive and sustained development of any morbid process in the eustachian tube, or in the tympanic cavity, or in the labyrinth, finally, there may develop alterations in intralabyrinthine pressure resulting in varying degrees of hearing failure which may be attended, finally, by one or more very distressing symptoms, as for example, vertigo, nausea, vomiting, tinnitus, nystgamus and disturbances of equilibrium and orientation. Kobrak advances the suggestion that vertigo following tympanic inflation, subjection to loud noises and massage of the drum membranes may be due to displacement of labyrinthine fluid by the strong contractions of the middle ear muscles producing a change in the position of the stapes. Such a condition continued over a long period of time would certainly produce impairment of the hearing.

In some cases of chronic middle ear suppuration a thin seropurulent discharge may be observed coming from an ear that is profoundly deaf. This is usually due to an ulcerative perforation in the membrane in the fenestra rotunda which permits leakage of labyrinthine fluid from the scala tympani and consequently reduction of labyrinthine pressure and interference with the transmission of

sound impulses to the membrana basilaris. In some of these cases the reduction of labyrinthine fluid pressure performs a more destructive role in the etiology of profound deafness than the infection and necrosis per se. The results observed by Culler, Finch and Girden[3] during the course of their animal experiments convincingly demonstrated that the function of the membrane in the fenestra rotunda is to compensate for and synchronize with pressure impulses transmitted to the labyrinth by stapedial oscillations. Therefore, a perforation in this membrane completely abolishes all physiologic function. Also, the function of this membrane is more or less completely abolished by an aggregation of inflammatory products which converts it into a rigid, unyielding fibrous mass, and places an additional burden on the aqueductus cochleæ. Ordinarily the capacity of the aqueduct is not adequate for the fulfillment of any very considerable vicarious function. Either perforation or fibrous fixation of the round membrane produces disharmony of the interdependent structures of the labyrinth and results in morbid changes in pressure and definite progression of hearing failure.

In an essay[1] presented in the Section on Ophthalmology and Otolaryngology at the St. Louis meeting of the Southern Medical Association I discussed several aural conditions coming within this classification and, considered in connection with the foregoing paragraphs of this article, I deem further elucidation here unnecessary. But there are other morbid conditions that belong to this group, two of which I will now consider.

ALLERGY

The tympanic cavity is subject to the same types of allergic reaction as are the paranasal sinuses or the nasal passages. Therefore, the middle ear may be regarded as an accessory nasal sinus when considered from a pathologic point of view. Though much further removed from the nasal passages it communicates with the nose just as freely as do the paranasal sinuses.

An apparently harmless substance, as for instance an ordinary article of diet, can, to an individual who is hypersensitive, become one of the serverest poisons that medical science has to encounter. Almost all of the tissues in the body of a person who has an allergic diathesis are affected in a manner that is distinctive and characteristic. Duke[5] points out that patients do not, as a rule, react to agents with which they frequently come in contact in large amounts. If highly sensitive persons were to come in contact with the exciting agents to which they react in gross amounts they would either succumb in death or acquire tolerance. Duke says:

"They react rather to substances which are rarely met with, or substances which are met with commonly but in almost infinitesimal amounts. * * * Patients who are sensitive to pollen, of course, meet with only traces of pollen under normal conditions. They do not meet with enough pollen to give them tolerance. * * * The infinitesimal trace of the substance which can cause reaction can hardly be measured. For example, in a pollen sensitive case, I have known one ten-thousandth of a milligram of crude pollen to cause reaction. If the active substance in pollen responsible for reaction could be purified and used, * * * the quantity which could cause reaction would be reduced to one one-millionth of a milligram or less—that is one one-billionth of a gram."

As a result of even mild allergic reaction, the tympanic mucosa becomes waterlogged and therefore greatly thickened, and transudation of serous fluid into the tympanic cavity quickly follows, hindering acoustic oscillations, causing increase in pressure and impairment of the hearing. In the presence of a severe allergic reaction this may occur quite suddenly and progress so rapidly that spontaneous rupture of the membrana tympani results in a very short time, turning loose a sterile serous fluid. According to Lewis,[6] who observed and reported on a group of cases of middle ear allergy, spontaneous rupture of the membrana tympani with a copious discharge of serous fluid was the rule, while Proetz[7] states that he did not observe an instance of spontaneous rupture in a series of cases that came under his observation. It is reasonable to assume that the apparent discrepancy in the occurrence of the phenomenon of spontaneous rupture in the reports of these observers was due to a difference in severity of the reaction in the two groups reported upon and not to an error in diagnosis, as the clinical manifestations in both groups clearly indicated the presence of middle ear allergy. If an individual is removed from contact with the agents to which he is hypersensitive, all the manifestations of allergic reaction rapidly disappear, provided permanent alterations have not already developed, and in cases of spontaneous rupture of the membrana tympani the perforation usually heals completely in from thirty-six to seventy-two hours.

I believe it is logical to assume that many of the acute exudative and recurrent types of catarrhal otitis media in persons who do not have a dominant allergic diathesis but who are more or less constantly exposed to agents to which they are mildly sensitive, or to agents to which they have acquired some degree of tolerance, are due to allergic reaction with transudation of fluid into the middle ear from pathologically permeable and engorged capillaries. Then it is reasonable to conclude also that the allergic process, in many instances, assumes a chronic form with consequent progressive hearing failure and the probability of developing vasomotor disturbances in the labyrinth. Injury to the terminals of the cochlear branch of the auditory

nerve in the hair cells of Corti's organ may ensue as a result of transudation into the labyrinthine spaces and finally result in profound deafness. Involvement of the canalicular and vestibular divisions of the labyrinth would almost certainly initiate the syndrome of Ménière or Lermoyez, especially the former. Since the perilymphatic spaces are continuous with the subarachnoid spaces it is reasonably certain that a prominent factor in the etiology of migraine may be accounted for on the basis of such an allergic process. The chronic cases are necessarily of the perennial type and may be due to the presence of either a foreign protein sensitivity or a bacterial sensitivity, or both. Usually in the mixed cases the sensitivity to foreign proteins is manifested first and bacterial allergy follows as a result of subsequent infection.

Ménière's syndrome is a rather frequent complication during the course of chronic aural allergic reaction, and is induced by transudation into the labyrinth producing a lesion that raises intralabyrinthine pressure and involves the vestibular nerve terminals in the utricular macula and the semicircular canals. It is justifiable to include allergy in the group of primary causes of Ménière's symptom complex. Duke[5] reported some characteristic cases of Ménière's syndrome caused by allergy and commented as follows:

"1. In each case the patient had the classical symptom complex known as Ménière's syndrome. 2. In each case the patient had a marked case of allergy. 3. In each case the Ménière complex was relieved by epinephrin. 4. In each case the Ménière complex was partly or wholly relieved over several weeks' trial by the avoidance of foods to which the patients gave positive intracutaneous tests. 5. In each case Ménière's syndrome was reproduced during a well period by the intracutaneous injection of extracts of the foods to which the patients showed skin hypersensitiveness and by the eating of such foods."

Allergic reactions may be divided into two general classes, namely, contact allergy, which includes all manifestations occurring as a result of direct contact of the allergen with a hypersensitive surface, and general allergy, which includes all manifestations occurring as the result of general reaction after the exciting agents have been absorbed from the gastrointestinal tract and distributed by the blood. Allergic affections of the ear are usually, though not always, local manifestations due to a general reaction in persons who have an allergic diathesis.

THE SYNDROME OF MÉNIÈRE

Since 1861, when Ménière, a Frenchman, gave to the medical world the classical description of the so-called disease that has borne his name, there has been much and varied speculation concerning the

etiology, and many recommendations have been advocated referable to the treatment of this symptom complex. Until recently the total accomplishment of these endeavors has been of little benefit to the patient. However, it seems that some progress has been made; but we have much to accomplish yet in the achievement of a definite knowledge of the varied pathologic changes responsible for the distressing symptoms comprised in Ménière's syndrome. Ménière believed the malady was due to a hemorrhagic exudate in the semicircular canals, and for a long time his opinion was generally accepted. So far as I am aware he did not attempt to explain the primary cause of the hemorrhage.

The syndrome of Ménière comprises characteristic and well defined clinical manifestations concerning which, it has been estimated, more than 300 articles have appeared in the literature on otology. In a typical case the seizure begins with a violent attack of vertigo in a person apparently well but with impaired hearing which is usually unilateral. Occasionally there may be bilateral impairment of cochlear function, but more profound in the ear to which the distressing tinnitus is referred. Nausea, vomiting and nystagmus are associated symptoms. The patient's face is pallid and bathed in clammy perspiration. He feels as if impending dissolution were drawing near, and he presents the pathetic aspect of absolute, helpless prostration. The seizure, if severe, may last for hours or even days, and pass off leaving the patient apparently well again until some time later when another attack follows. Life, then, is a succession of such seizures occurring at irregular intervals, and deafness advances in a slow and irregular but definitely progressive course.

Many theories have been advanced to explain the pathologic changes that may give rise to this syndrome, but it has not yet been wholly explained, and many features of the affliction still remain concealed in mystery. But few observers now believe, as Ménière did, that the syndrome is dependent upon an intralabyrinthine hemorrhage, except in rare instances, and even then that there is always an antecedent pathologic change. To those who believe the pathologic change may be located primarily in the labyrinth it may be pointed out that clinical statistics show that in a few cases even destruction of the labyrinth failed to give relief. To those who hold that the pathologic alterations are usually in the acoustic nerve attention may be directed to the fact that in some instances microscopic examination of sections of this nerve fails to reveal morbid changes in the specimens submitted for study. Furstenberg, Lashmet and Lathrop[9] call attention to an interesting discovery made by Lowenburg, a pathologist, in his study of a fatal case of tumor of the acoustic nerve in which he found

a normal nucleus of a completely degenerated auditory nerve. They advance the suggestion that this phenomenon may indicate innervation of the nucleus from the opposite side, and that this may explain the failure, in some instances, to obtain relief from the dominant symptoms following section of the acoustic nerve.

It may be concluded, therefore, that it is not practicable definitely to ascribe Ménière's syndrome to a lesion in any particular part of the labyrinth or the auditory tract. It would appear more reasonable to assume that any or all of the labyrinthine structures or the auditory tract, including the nucleus, may be involved in morbid alterations which give rise to an overwhelming irritation of the vestibular nerve terminals in the utricular macula and the semicircular canals. Also, there is good reason for the conclusion that Ménière's syndrome is always caused by an increase in intralabyrinthine pressure, whether gradual or sudden, which is the result of either local or systemic pathologic alterations that excite morbid stimulation in the vestibular nerve terminals. This conclusion is well supported by the experimental and clinical observations of several investigators. Dohlman[10] demonstrated that nystagmus occurs only as a result of bending the cupola in one direction or the other. Bending the cupola can result only from an increased intensity of either an ampulla-efferent or an ampulla-afferent endolymphatic flow. The technic of Dohlman's experiments was very similar to the method devised by Steinhausen and confirmed that observer's conclusions. Steinhausen[11] demonstrated that a flow of endolymph in the semicircular canals is started by ordinary movements of the head, and at the same time the cupola terminalis changes its position, and this change produces stimulation in the vestibular nerve terminals in the sensory epithelium. Apparently the experiments of both Dohlman and Steinhausen satisfactorily explain the modus operandi of stimulation, whether physiologic or morbid, in the semicircular canals. Wittmaack[12] conducted similar experiments, and his conclusions, while somewhat different from those of either Dohlman or Steinhausen, for practical application mean substantially the same as theirs. He observed that only a rapidly increasing intensity of peripheral pressure at the cupola can be specific for a stimulation receptor, and he believes, therefore, that on this basis all pathologic types of nystagmus can be explained and, consequently, that the nystagmus of Ménière's syndrome belongs in this group and is caused by an increase in pressure of the perilymph. Thus Wittmaack believes that Ménière's syndrome is caused by a rapid increase in intralabyrinthine pressure which is superinduced by a temporary increase of fluid within the peripheral labyrinthine spaces.

Foldes[13] believes that the systemic and local retention of water, sodium salts and other mineral substances frequently produces disease in one or more organs and performs a distinctive role in the pathogenesis of epilepsy, migraine, eclampsia of pregnancy, angina pectoris, bronchial asthma and allergic reactions. He believes that Ménière's syndrome belongs in this group. In corroboration of his conviction Foldes refers to the results reported by Dida Dederding of Copenhagen who, proceeding on the assumption that this syndrome develops as a result of an edema in the labyrinth, found that there was a marked improvement following diuresis by the administration of salyrgan, and that there was invariably a relapse of the Ménière manifestations following a repetition of water retention.

For the treatment of the diseases belonging in this group Foldes developed an antiretentional therapy and diet regulation which appear to have considerable merit. He reported some patients treated by his method who had suffered from tinnitus and deafness and weekly attacks of dizziness, nausea and vomiting, and stated that in each instance, within a few days after beginning the treatment, the attacks ceased completely.

Because of the existing speculation and confusion concerning the etiology of Ménière's syndrome, and because of the inability to relieve victims of the malady by the hitherto empiric methods of treatment, and because of the encouraging success reported by Dederding in the treatment of patients by a process of dehydration, Furstenberg, Lashmet and Lathrop[9] undertook to verify the observations of Dederding. It was soon apparent that the method of treatment advocated by Dederding was neither sufficiently comprehensive nor accurate in detail to merit general clinical application. Therefore, they found it expedient to enlarge the scope of their investigations and clinal experiments. The first patient selected for study had been afflicted with typical Ménière's disease for ten years. For two years he had had an average of two or three attacks weekly, and for six months he had had daily attacks. All the symptoms were characteristic and severe. The patient was allowed a measured diet and fluid intake. At the end of the second day there was considerable water retention, half of which was lost during the three succeeding days, and there were no attacks of vertigo. Then for two days he was given additional sodium chloride in his diet, which was soon followed by increased water retention and a severe return of all symptoms. Then the patient was dehydrated for three days by a minimal allowance of water intake and was given sodium bicarbonate. Although the systemic content of water was low, after the sodium was administered he had another severe seizure. Then sodium was re-

stricted to what is necessary in the constant diet and ammonium chloride, an acid-producing salt, was administered in ascending doses. This apparently promoted elimination of sodium from the system and the attacks ceased immediately, regardless of water retention. The authors concluded that Ménière's syndrome may be due to an excess of sodium salts, particularly the chloride, which is known to cause systemic water retention, and which may cause edema of the labyrinthine structures and increased pressure.

On the basis of this conclusion fourteen cases of characteristic and uncomplicated Ménière's syndrome were treated successfully by the method devised by the authors. In no instance was there a failure to induce an attack by the administration of sodium; and in no instance was there a failure to secure complete relief by restricting sodium intake and the administration of ammonium chloride. Water was allowed freely, as the authors found that water had nothing to do with the condition so long as a reserve of sodium chloride was not accumulated. It is well known that nephritic patients can not tolerate sodium chloride, except in minimal amounts, because of the edema it produces in the kidneys. Apparently the effect produced by sodium chloride in the labyrinth of a susceptible individual is analogous to the effect it produces in the kidneys of a nephritic patient.

In the treatment of a few cases I have verified the results reported by Furstenberg and his associates. However, I have found it necessary to be very cautious in approaching the huge dosage of ammonium chloride recommended by these authors. It should be remembered that this drug is an acid-producing salt, and if pushed vigorously without due caution it may impair the digestion, injure the structure of the red blood cells, increase tissue waste and tend to induce chloropenia and ketosis, particularly if the intake of the chloride and bicarbonate of sodium is limited to only the bare amount of the patient's systemic requirement, or less, thus producing a symptom complex simulating very closely, if not actually constituting, the acidosis syndrome. I have seen this occur as a result of smaller dosage than these authors recommend and continued for several days.

Portmann[11] believes that superinduced and irregularly passing periods of hyperemia of the labyrinth are caused by impaired tonicity of the vasosympathetic apparatus resulting from some systemic disturbance that produces increased pressure. He devised an operation for the purpose of reducing pressure by entering the labyrinth through the mastoid process and making an opening in the saccus endolymphaticus, thus reducing pressure by draining off lymphatic

fluid. McNally[15] criticised Portmann's operation on the ground that the opening in the saccus soon heals over completely and therefore no permanent relief reasonably could be expected.

If Portmann's concept of the morbid process is the correct view, finally, in any case, gross vascular changes with a sclerotic pathology and a consequent liability to hemorrhage may ensue, and the patient would be in danger of hemorrhage from the intracerebral vessels provoked by repeated seizures of Ménière's syndrome. That this is true in some instances there can be no reasonable doubt, as it is a matter of common experience to observe patients suffering from vascular hypertension who have a feeling of fullness in the head, vertigo, nausea, tinnitus and dullness of hearing. Also hypersensitive patients who suffer from these aural manifestations are occasionally stricken down with cerebral apoplexy.

Since it is reasonably certain that the saccus endolymphaticus is an absorption chamber for the disposal of excess endolymph and for maintaining a constant physiologic endolymphatic pressure, logically it may be assumed that either edema or hyperemia of the labyrinthine structures would more or less completely abolish absorption of endolymph and tend to increase pressure. Also, edema or hyperemia in the labyrinth would involve the aqueductus cochleæ and retard or completely block the flow of perilymph into the cranial cavity, and raise intralabyrinthine pressure. It is probably true that suppression or abolishment of function of the saccus endolymphaticus and of the aqueductus cochleæ may be the dominant factor in the morbid process in the labyrinth that produces the Ménière syndrome.

A variety of systemic causes have been mentioned as factors in the etiology of Ménière's syndrome. Among these are allergic reaction, heat stroke, pernicious anemia, nephritis, syphilis, vascular hypertension, the leukemias, diseases of the central nervous and vascular systems, focal infections and toxemia and such exogenous poisons as lead, alcohol and tobacco. These are mentioned because of the frequency of the concurrent initiation of Ménière's syndrome in the course of any one or a combination of two or more of these diseases. Therefore, the so-called Ménière's disease is not a separate and definitive disease entity, but is a complex of symptoms arising from widely variant and scattered morbid alterations.

SUMMARY

1. Normal hearing is dependent upon rhythmic, physiologic changes in the intensity of intralabyrinthine pressure between the windows.

2. There is evidence to show that morbid changes in intratympanic pressure, either negative or positive, may extend to the labyrinth and produce impairment of hearing.

3. Chronic middle ear suppuration may cause serofibrinous exudation in the labyrinth and produce deafness or seropurulent infection and endanger life. Also, chronic suppurative processes in the middle ear may perforate the membrane in the round window, give rise to drainage from the scala tympani and result in reduction of pressure and destruction of hearing.

4. Allergic reaction in the tympanic cavity is comparatively frequent, and if acute may cause temporary damage, or if chronic, permanent damage of cochlear function, and is an important factor in the etiology of Ménière's syndrome.

5. There is evidence to show that Ménière's syndrome is always the result of an increase in intralabyrinthine pressure resulting from a variety of either local or systemic morbid processes.

6. There is evidence to show that patients suffering from Ménière's syndrome have but little tolerance for sodium salts, particularly sodium chloride. Therefore they should have only enough sodium chloride to preserve the isotonicity of the body fluids and no more.

7. There is evidence to show that to designate Ménière's syndrome as a definitive disease entity is a misnomer. It is a reflex symptom complex resulting from morbid processes that secondarily affect the soft structures of the internal ear and produce an increase in intralabyrinthine pressure.

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Clinical Notes

XLVI

AN UNUSUAL MENINGO-ENCEPHALOCELE ETHMOIDALIS: REPORT OF A CASE AND REVIEW OF THE LITERATURE

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REPORT OF A CASE

CASE 1.—R. L., a white male, 2½ months of age, was referred by Dr. McClure because of a tumor on the dorsum of the nose. Following a moderately prolonged labor, the mother had been delivered of this baby by a low-forceps operation without difficulty. The tumor was noted at birth, but was thought most likely to be a hematoma. The baby was normal in every other respect. The parents stated that the tumescence had not changed size from birth.

Examination revealed a round, smooth, slightly movable mass the size of an olive, involving the left bridge of the nose and encroaching upon the mesial canthus of the eye. It had a semi-firm consistency, although one spot appeared to be quite hard. The overlying skin was not reddened and could be slightly moved. No pedicle or skull defect could be palpated. Light was not transmitted. The size and shape of the swelling was unaffected by crying or coughing and it could not be reduced by manipulation. Roentgenograms of the skull did not reveal a defect in the skull.

A tentative diagnosis was made of a teratoma, and after a few weeks of observation, during which time the mass did not change size, operative removal was advised.

The operation was performed under ether anesthesia. A curvilinear incision was made through the skin, but no definite capsule could be found. Sharp dissection was necessary throughout the operation, due to the dense connective tissue surrounding the tumor. The mass was intimately associated with the lateral nasal cartilage, and difficulty was had in freeing this structure. A careful search failed to reveal either a pedicle or a cranial defect. The recovery was uneventful, and an excellent cosmetic result was obtained.

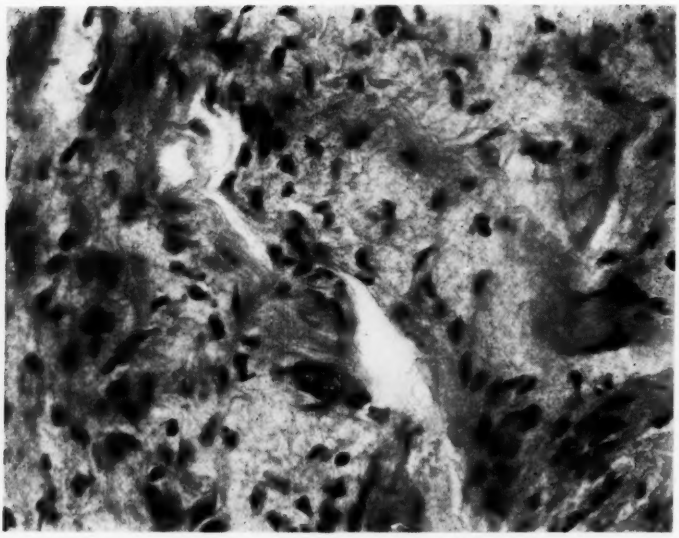

Fig. 1. A high power area showing the intermingling of fibrous tissue, which is representative of the meninges and the neuroglia tissue, which is representative of the brain. (440 magnification.)

The tissue removed was examined by Dr. C. V. Weller, Professor of Pathology at the University of Michigan, who submitted the following report: "This shows a mixture of atypical neuroglia containing a few poorly developed ganglion cells and dense connective tissue. This is a developmental disturbance. The situation described shows it to be a meningo-encephalocele ethmoidalis. We have seen several of these before, but almost always from newborn infants and as examples showing communication with the ventricular system of the brain. I would not be surprised if x-ray study of the base of the skull shows a bony defect in the ethmoid region."

After a review of the literature pertaining to developmental disturbances whereby brain substance or its coverings become extracranial, it was learned that no one theory as to the etiology has been absolutely accepted.

Lyssenkow,[1] in 1896, classified cranial congenital malformations into two groups, the exencephaly and the cephalocele. He stated that in the former group a general malformation of the skull was present,

while in the latter group there was only a partial developmental failure.

Lampert[2] defined exencephaly as being a larger or smaller portion of the brain substance which had been dislocated through an opening into the cranium. He stated that it might be looked upon as an ectopy of the brain and was of but teratologic interest, as it was incompatible with life.

As to the etiology of this class of disturbances, Lampert states "occlusion of the medullary tube begins in the mesencephalon (middle cerebral ventricle), extending caudad and cephalad; the frontal and occipital portions, however, close much later. Therefore, there are greater chances for abnormalities to take place, such as delayed or incomplete fusion of the medullary tube, abnormal growth of brain tissue, with ultimate formation of cerebral herniæ. A similar explanation, delayed closure, holds good also for the cases of occipital herniæ." Mittendorf,[3] reporting a series of 93 encephalomeningoceles, cites 16 as having a frontonasal origin.

W. P. Haggard[4] sums up the etiologic factors which are thought to bring on these congenital anomalies thus:

"1. Union of brain sac with amnion interfering with the development of bone (Funkhouse[5]);

2. Disease of bone, causing non-union or porosities, with protrusion of brain covering through the aperture, probably coexisting with disease of the brain itself;

3. Intrauterine hydrocephalus with bulging as the bones come together (Gross[6]); and

4. Exostoses or protuberances on the pelvis of the mother, upon which the fetal head rests, interfering with development."

It is the author's opinion that this particular case exemplifies the type discussed by Gross. It seems logical to assume that a hydrocephalus was present, due to a temporary malformation of the developing medullary tube, whereby a portion of the brain was displaced cephalad. By the time the cranial bones were developed in this region, the malformation had ceased to exist, the internal pressure had disappeared, and the brain was forced to sacrifice the herniated portion because it was pinched off by the development of the skull.

CONCLUSIONS

A brief summary is given of the various theories as to the cause of congenital extra-cranial brain substance, and an unusual case is

cited which best seems to satisfy the postulates given by Gross, who believed that the immediate pathogenesis of cephalocele was that a primary malformation resulted in an intrauterine hydrocephalus.

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XLVII

PEDUNCULATED FIBROMA OF THE NASAL SEPTUM*

W. Likely Simpson, M.D.†
AND
J. D. Evans, M.D.†

Memphis

Benign tumors of the septum, in the writers' experience, are quite a rare condition. Granulomas, telangiectatic polypi and angiomas are more frequently seen than myxomas, fibromas and chondromas.

ETIOLOGY

Usually no definite etiologic factor can be found which will account for all these tumors, but trauma, chronic irritation and sinus infection have been given as causes.

LOCATION

It seems to us that granulomas, angiomas and fibromas are usually found in the anterior half of the septum, while myxomas are in the middle or posterior third, and chondromas, as described by Walter Howarth,[1] may be found in any part of the septum.

REPORT OF A CASE

Case 1. Dorothy B., negro, came complaining of an obstruction of the left side of her nose for the past two months—the obstruction gradually becoming larger—with a history of slight bleeding. There was a history of epilepsy and a diagnosis of positive brain tumor and a pregnancy of five months' duration.

On examination, the left side of her nose was found to be practically closed by a firm mass about the size of a large hazelnut, with the color of normal mucous membrane. The tumor had a fairly wide base—about one-half the diameter of the tumor itself. The attachment was about one-half inch from the anterior naris. Manipulations did not produce any bleeding.

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*From the Department of Otolaryngology of the University of Tennessee.
†Presented before the Memphis Eye, Ear, Nose and Throat Society, spring, 1936.

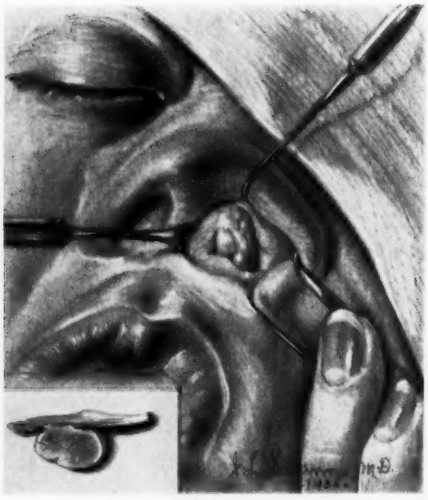

Fig. 1. Pedunculated fibroma of the nasal septum. Insert shows tumor on section.

Fig. 2. Anterior-posterior section through septum and tumor.

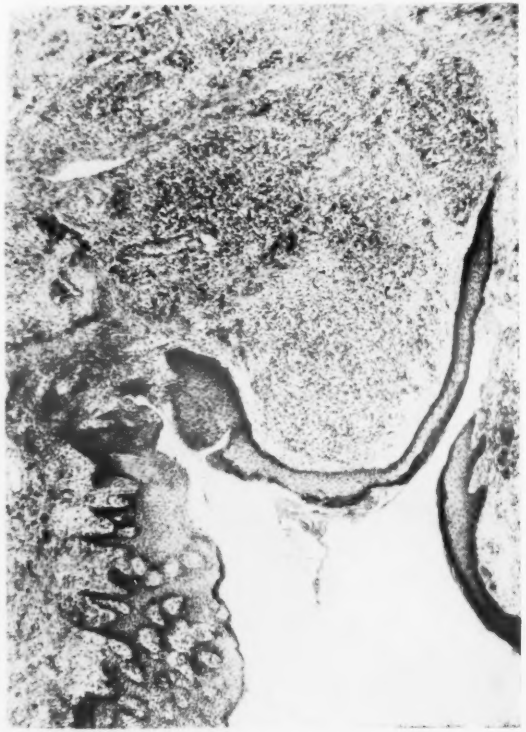

Fig. 3. Antertior portion of septal mucous membrane and tumor. First composed of fibrous tissue; second, of cellular fibrous tissue, both covered by stratified squamous epithelium.

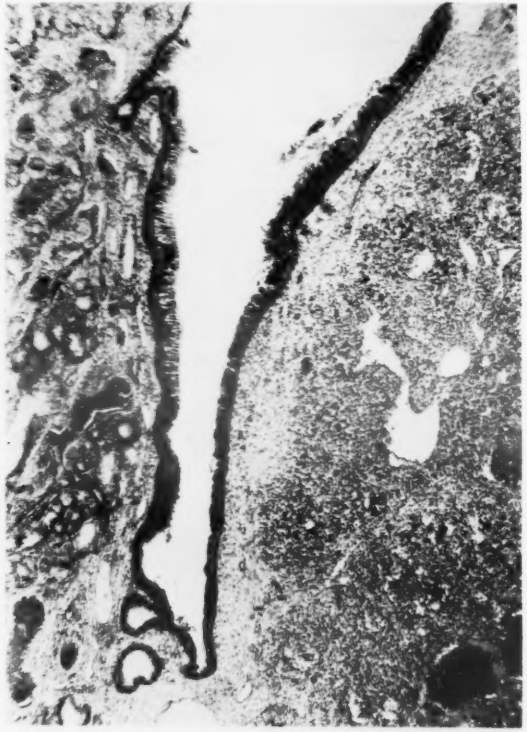

Fig. 4. Posterior portion of septal mucous membrane and tumor. First composed of fibrous tissue and mucous gland; second, of cellular fibrous tissue rich in dilated blood vessels, both covered by pseudostratified ciliated columnar epithelium.

Under local anesthesia the growth was removed with the mucous membrane of one side of the septum and the entire thickness of the cartilage. A sliding flap of the remaining mucous membrane on the same side of the nose was done. An uneventful recovery was made.

Examination of the specimen of the tumor revealed the following:

Macroscopic: The specimen consisted of a lobulated, pink, moderately translucent, soft mass attached by a small stalk to the mucous membrane, covering a piece of cartilaginous septum. The mass measured 1 x 1 x 0.5 cm.

Microscopic: The cartilage was covered on one surface by mucous membrane, which anteriorly to the polyp consisted of fibrous tissue covered by stratified squamous epithelium, while posteriorly to the polyp it consisted of fibrous tissue containing mucous glands and ducts and was covered by pseudostratified columnar epithelium. The polyp was composed of a cellular fibrous tissue rich in dilated blood vessels and over its anterior portion was covered with stratified columnar epithelium wihch was continuous with that of the posterior part of the septal mucous membrane.

Diagnosis: Pedunculated fibroma of the septum.

We are indebted for the pathologic report of this specimen to Dr. Harry C. Schmeisser and for the photographs to Dr. Joseph L. Scianni, Director and Artist, respectively, of the University of Tennessee Pathological Institute.

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XLVIII

SUBMUCOUS CORDECTOMY FOR BILATERAL ABDUCTOR PARALYSIS: REPORT OF A CASE*

WALTER STEVENSON, M.D.

QUINCY, ILL.

Bilateral paralysis of the abductor muscles of the larynx, though not common, is one of the most troublesome and distressing conditions with which the laryngologist must contend. When it occurs on the operating table immediately following thyroidectomy, it constitutes a real emergency. It has been said that when unilateral paralysis follows thyroidectomy, it is a misfortune, but when bilateral paralysis ensues it is a real tragedy.

Many methods for increasing the size of the glottis for relief of this condition have been proposed; the multiplicity of operations suggested apparently indicates the inadequacy of end results. Chevalier Jackson stated that endoscopic evisceration was successful in but one case. He[1] later suggested ventriculocordectomy, reporting almost 50 per cent successful results, though he warned that the operation must be limited to those cases entirely free of cicatricial stenosis. I have tried this operation without success, due to subsequent cicatricial contractures.

At the 1931 meeting of this Society, Hoover[2] reviewed the condition and reported four cases relieved of stridor, and with good phonation following submucous resection of the cords. He stated that a number of such operations had been done by Killian, Dahmann, Weingarten and Albrecht, with very promising results. In this article is a complete review of the literature, together with the results of methods tried in the Lahey Clinic and his conclusion that submucous resection of the cords is a relatively safe and certain method of overcoming the obstruction of bilateral abductor paralysis.

Recently Lore has modified this operation, stating that his method was based on anatomic and physiologic reasoning. The

*Presented before the combined meeting of the Middle and Midwestern Sections of the American Laryngological, Rhinological and Otological Society, Chicago, Monday, January 11, 1937.

principles involved in his operation are the same as Hoover's, but should be more successful because of a greater glottic width resulting from total ablation of the arytenoids. Ideal treatment of bilateral abductor paralysis would be end to end suture of the injured nerves or anastomosis with other functioning nerves, thus restoring physiologic action of the paralyzed muscles. This has been tried with but mediocre success. I quote from a personal communication by one of the best known neurologic surgeons in this country: "The operation devised by Frazier for trying to pick up the recurrent laryngeal nerves for bilateral abductor paralysis is a frightful job; I have tried it once and failed, and I think the cases that Dr. Frazier reported are nothing to boast of. The nerves are so small it is almost impossible to find them in the scar of a thyroidectomy wound. I can't help feeling that your suggestion of doing a cordectomy is the wiser plan. The case I tried to do—I swore I would never try another one again."[4]

Theoretically if Semon's law is more than a hypothesis, then section of each recurrent nerve should place the cords in the cadaveric position with good airway, though with much damage to the voice. Practically, such a procedure has never been successful.

It is interesting to note that the history of my case in its early stages followed the postulates of Semon's hypothesis, although the final position of the cords does not support the theory.

REPORT OF A CASE

CASE 1.—Miss A. W., aged 17, entered the hospital October 9, 1932, for a subtotal thyroidectomy. Other than hyperthyroidism, the general physical examination was negative. Operation was done on October 19, 1932; as the wound was being closed it was noted that the patient had some huskiness of voice, and was having considerable inspiratory stridor. After she had been returned to bed, I saw her in consultation. Both cords were in the paramedian position with only slight movement on phonation and respiration. Several visits were made that day, and because of increasing inspiratory distress the wound was reopened and tracheotomy done. An uneventful convalescence ensued and one week following the tracheotomy the cords were in cadaveric position; there was no respiratory distress when the tube was stoppered, but phonation was reduced to a whisper. The patient was decannulated on the eighth day and a week later there was definite movement of the left cord in phonation. This was followed by almost complete recovery of phonatory and respiratory movements in each cord.

I saw the patient again on March 21, 1933, when she gave the following history of the intervening period. After discharge from the hospital she breathed and phonated comfortably and easily until six weeks before this observation. Then inspiration had become increasingly difficult, worse at night, so much so that when asleep she made a loud crowing noise which awakened everyone in the home. At this time phonation was impaired, although a good voice was present. Examination now, six months after operation on the thyroid, showed the cords in midline position, ballooning upward on expiration and nearly approximating during inspiration. Sedatives were given because of extreme nervous apprehension. She was placed in the hospital for observation. After two weeks it was concluded that she had sufficient airway to resume a restricted activity.

Following her release from the hospital there was a loss of forty pounds in body weight, and the patient was living in terror of suffocation. She was readmitted to the hospital on September 18, 1933, demanding relief from respiratory distress. On September 26, 1933, a tracheal tube was reinserted and the patient discharged from the hospital a few days later. She soon regained the lost weight and seemed happy with the tracheal tube, closing its opening for phonation. However, she gradually became dissatisfied and discouraged because of the cannula and finally demanded that something be done to relieve her of the tube, even though she had been told that removal of her cords would probably reduce her voice to a whisper. On October 10, 1934, I did a submucous cordectomy, according to the method described by Hoover, under local anesthesia, which is quite adequate. This operation is not so simple as his article would indicate, though easy to do on a cadaver. His suggestion that coagulation controls the small amount of bleeding was not my experience, since hemorrhage from the submucous pockets was copious and difficult to control, and hemostasis became complete only after tightly packing the larynx. The cartilage and internal structures were sutured and the end of the packing brought out through the cricothyroid membrane. A part of the packing was removed each day, and the remainder removed on the fourth day. On the fifth day the opening in the cricothyroid membrane was closed and the patient discharged. Twelve days after operation the patient returned to the hospital almost exsanguinated because of a severe secondary hemorrhage from the larynx, caused by a severe strain of the voice in a fit of anger. A longer period of complete rest would have prevented this accident. The wound was reopened and repacked by a colleague during my absence. The pack was removed on the fourth day, the tracheal cannula was re-

moved one week later, and the patient made an uneventful recovery. At this time the patient was contented, had ample airway and had developed a voice of fair strength and quality. She was last seen on November 6, 1935, thirteen months after my submucous cordectomy, at which time she was still comfortable and had a fair voice; that day she was struck and instantly killed by an automobile.

COMMENT

A case of postoperative bilateral abductor paralysis is reported. There was spontaneous recovery within ten days. Subsequently, a complete total paralysis developed, which was relieved by submucous resection of the cords.

Objection could be made that a longer period of observation might have altered my conclusion regarding the method of operation. I feel that the elapsed time of thirteen months without change in the end result probably indicated permanent cure.

My experience leads me to believe that submucous cordectomy, as described by Hoover, is a satisfactory procedure.

908 W. C. U. Bldg.

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XLIX

UNUSUALLY LARGE FOREIGN BODIES REMOVED FROM THE TRACHEOBRONCHIAL TREE*

MURDOCK EQUEN, M.D.,

AND

FRANK NEUFFER, M.D.

ATLANTA

So much has been said and written in recent years about the early diagnosis of malignant tumors of the larynx, tracheobronchial tree and esophagus, the treatment of cardiospasm, bronchial stenosis, lung abscess and bronchiectasis, and even the investigation of asthma, that we tend to minimize the importance of the original use of the bronchoscope in the removal of foreign bodies. It is, however, worth while to bring up again the removal of foreign bodies unusual in size or nature, or unusual in difficulties of diagnosis or in complications.

REPORT OF CASES—GROUP I

CASE 1.—A boy of 10 was playing with some upholstery brads in his mouth. With sudden laughter one was aspirated into the right main bronchus. Although several weeks later the x-ray indicated extensive involvement of the lower lobe, bronchoscopic removal of the brad was followed by prompt recovery.

CASE 2.—A girl of 10 aspirated a pencil sharpener two days before entering the Infirmary. The x-ray showed that the sharpener had entered the right main bronchus, the small end first, and it was thus acting as a plug. She left the Infirmary well, nine days after bronchoscopic removal.

CASE 3.—A college girl with several hairpins in her mouth laughed suddenly and aspirated a bobby pin 5 cm. in length. The x-ray showed it in the right main bronchus. The removal of this hairpin on the third day was a simple procedure.

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*Presented before the Southern Section of the American Laryngological, Rhinological and Otological Society, in Memphis, on January 13, 1937.

Ponce de Leon Eye, Ear and Throat Infirmary.

Fig. 1. Anteroposterior view of upholstery brad in the right main bronchus. Note the involvement of the lower lobe. (Case 1.)

Fig. 2. Case 1. Lateral view.

Fig. 3. Anteroposterior view of pencil sharpener plugging the right main bronchus. (Case 2.)

Fig. 4. Case 2. Lateral view.

CASE 4.—A case of peculiar interest we think is that of a boy of 12. This youngster entertained himself at school shooting needles through a blow-gun. A bit of cotton attached to the needle served both to allow him to get force behind the needle and to steady the missile in flight. He boasted that he could annoy his schoolmates twelve feet away. One day, taking in too deep a breath preparatory to a shot, he aspirated both the needle and the cotton. An x-ray at Fort McPherson showed the presence of the needle in the trachea above the bifurcation. He was referred to the Infirmary two hours after the accident. As the needle was withdrawn it became separated from the cotton wad; the cotton was recovered by introducing the forceps a second time. Three days later the boy was well.

CASE 5.—A lecturer, aged 41, was putting up some charts, holding tacks in his mouth. After an unexpected cough he aspirated three tacks. He was sure that he had "swallowed" these tacks and immediately consulted a doctor. As too often happens, he told him to forget it. Two days later, while lecturing, he noted wheezing on expiration, so he consulted a second physician, who advised an x-ray. This revealed three tacks at the bifurcation of the right main bronchus. With the aid of a biplane fluoroscope these were removed without difficulty and he was discharged from the Infirmary on the third day.

CASE 6.—A child of 10, in some extraordinary manner, managed to aspirate a corsage pin 8 cm. long. The point of the pin was upward and had perforated the wall of the bronchus and the head was out of sight in the bronchus to the lower lobe. Efforts to dislodge the head of the pin head downward in order to extract it point first through the bronchoscope were unsuccessful. The smallest pin cutter made was too large to go through a No. 5 bronchoscope. The child's head, therefore, was held perfectly still as the bronchoscope was withdrawn, and under the guidance of the biplane fluoroscope the pin cutter was immediately passed into the bronchus to grasp the pin. On cutting the shaft of the pin, its large head became loosened and slipped farther down into the lower lobe. The middle portion of the pin was removed with the pin cutter. The bronchoscope was reinserted and the pointed part of the pin was removed with forceps. With a bead forceps and fluoroscopic guidance, the head of the pin was recovered a few minutes later. Convalescence was uneventful.

COMMENT

This group of cases serves once again to illustrate how well the tracheobronchial mucosa tolerates a metallic object and how promptly

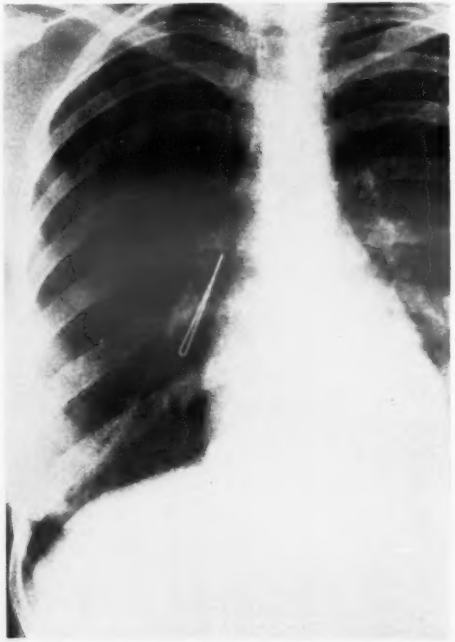

Fig. 5. Anteroposterior view of hairpin in the right main bronchus of a college girl. Note that the lung shows no reaction. (Case 3.)

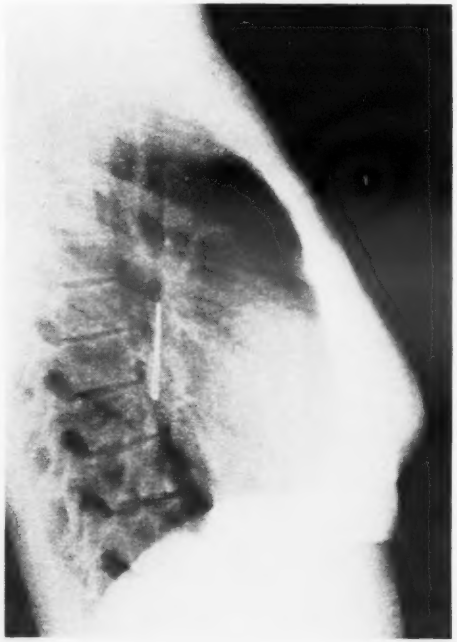

Fig. 6. Case 3. Lateral view.

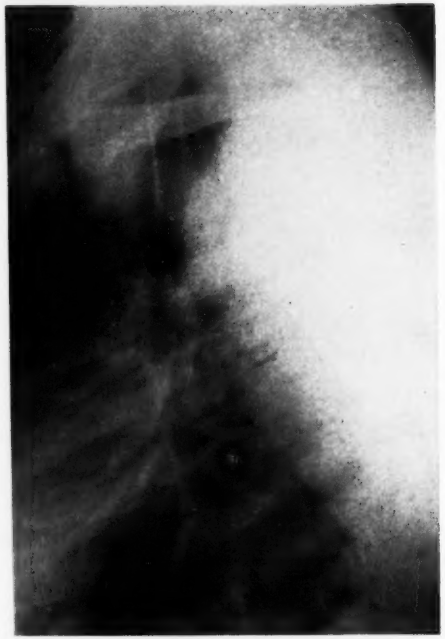

Fig. 7. Oblique view of a needle in the trachea of a schoolboy. The cotton wad below the needle casts no shadow. (Case 4.)

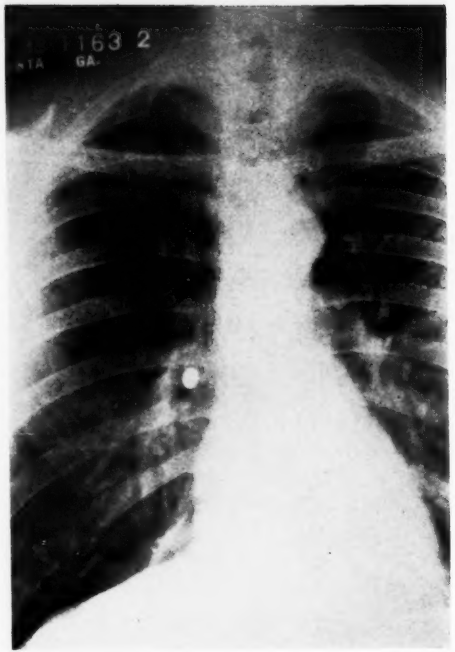

Fig. 8. Anteroposterior view of three thumb tacks in the right main bronchus of a man of 41. (Case 5.)

recovery may be expected to follow its removal. The first three are of interest principally on account of the size of the foreign body. Case 4 emphasizes that direct vision is desirable, because x-ray would not have revealed the wad of cotton. In Cases 5 and 6 the biplane fluoroscope was essential.

REPORT OF CASES—GROUP 2

CASE 7.—When a boy, aged 3, failed to recover from pneumonia of the right lower lobe, an x-ray of the chest was taken for diagnosis. This revealed a crooked sixpenny nail in the trachea with its head in the right main bronchus. After removing the nail, the sputum became slightly blood tinged, thick, yellow and profuse. On the fourth day a tracheotomy tube was inserted. Through this mucopus was aspirated as often as necessary. With the aid of three transfusions the child recovered three months after the operation.

CASE 8.—A girl of 3 told her parents that she had "swallowed" a nail, but her story was not believed. After four months of chronic illness with paroxysms of coughing and foul, bloody, mucopurulent sputum she was brought to Atlanta. X-ray revealed a sixpenny nail which was removed without difficulty. In spite of the use of carbon dioxide the lower lobe remained atelectatic. We performed tracheotomy for aspiration on account of profuse suppuration. The improvement was immediate and eight days later she was discharged cured.

CASE 9.—A girl of 10 aspirated a large bead. This would occlude first one and then the other main bronchus and produce symptoms in first one lung and then the other. With each cough this bead would be forced up into the larynx. Two days later she was referred to us. The mechanical trauma of the cough forcing the bead into the larynx and against the lower surface of the cords had produced marked edema. On account of this we found it necessary to bring the bead into the upper trachea and remove it through an incision. Although pneumonia developed, with a temperature of 106, the child was well in three weeks.

CASE 10.—A child, aged 2, was brought to the Infirmary on the third day after aspirating a navy bean. After the removal of the bean we hoped for an uneventful recovery, but because of profuse suppuration it was necessary to institute intratracheal drainage. On the twenty-second day the child was able to go home.

CASE 11.—A child of 6 choked, strangled and vomited immediately after "swallowing" half the shell of a prune seed. Later that day she began to have fever and to wheeze on breathing. After eight

Fig. 9. Anteroposterior view of an 8 cm. corsage pin in the bronchus of a girl of 10. (Case 6.)

Fig. 10. Lateral view of same.

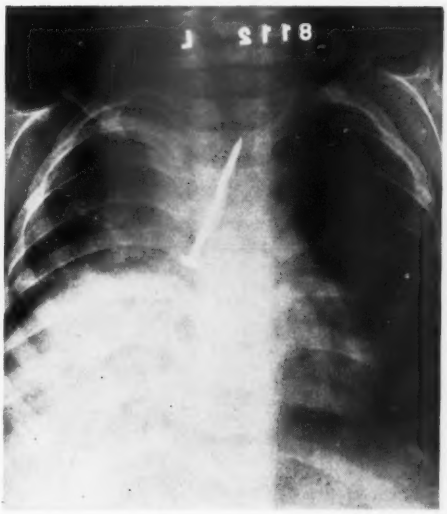

Fig. 11. Anteroposterior view of a sixpenny nail in the trachea and bronchus of a child of 3. Note the extreme involvement of the right lower lobe after the nail had been present 3 months. (Case 7.)

weeks of illness, with a high fever and profuse purulent sputum, she was brought to Atlanta. Intratracheal drainage was instituted. Although, of course, prune seeds are not opaque to the x-ray, the shifting of the pulmonary involvement convinced us that the foreign body was still present. On passing the bronchoscope considerable pus was sucked out and the half prune seed was located and removed. Improvement was dramatic. Thirty days later she went home well.

CASE 12.—The most unusual foreign body of all was a large piece of peach peeling aspirated by a girl of 4. She was brought in as an emergency case, deeply cyanotic, almost pulseless, suffering with extreme dyspnea. It was necessary to do immediate tracheotomy to relieve the mechanical obstruction. Through the incision the peeling was quickly removed from the upper part of the trachea. Profuse suppurative tracheobronchitis ensued. Suction through a small rubber catheter inserted in the tracheotomy tube was instituted. Ten days later the tube was removed and two weeks after the operation the child was allowed to go home.

COMMENT

Cases 7, 8 and 11 have been reported before, but, since they illustrate so well the principle of intratracheal drainage emphasized

Fig. 12. Anteroposterior view of a sixpenny nail in the trachea and bronchus of a child of 3. (Case 8.) There is much less consolidation in the right lower lobe than in Case 7.

Fig. 13. Lateral view of same.

by one of us four years ago, it is permissible to bring them up again. In pulmonary suppuration, as elsewhere in the body, pus must be evacuated, and when the body is unable to handle it, the surgeon must help. Increasing experience has more than ever convinced us that it is much safer to provide drainage through intratracheal suction than by repeated bronchoscopic treatments. For this purpose we use a small catheter. The closed end is removed and the rubber is softened in a match flame and its edges smoothed with gauze. As often as necessary the modified catheter can be passed through the tracheotomy tube as far down as possible and then connected to a suction apparatus for the aspiration of secretions. It is a simple procedure, one that can easily be executed by the nurse and one that does not cause the patient inconvenience, alarm or pain. It is also our custom frequently to instil 10 per cent caroid in mineral oil to prevent scabbing and crusting. Sometimes we also use benzoin inhalations, and in certain cases the oxygen tent has seemed to be a life-saving measure.

Cases 7 and 8 illustrate the final results of a large foreign body, even of metal, being allowed to remain in the bronchus.

Case 9 is the first in this series in which the foreign body was a "floater." Although it has often been said that an object small enough to get into the windpipe can also be removed through the natural channels, this bead proved the exception on account of the edema below the cords from the trauma of impact. It is well to remember that one can open the trachea for the direct removal of foreign bodies. At the same time, of course, this bead would have been difficult to remove without first having brought it up into the trachea with bronchoscopic forceps.

The navy bean in Case 10 was another "floater."

Case 11 was the third "floater" in this series. Case 12 presented a true emergency, and immediate tracheotomy was necessary to relieve the mechanical obstruction, which was caused not only by the size of the peach peeling but also by the extreme edema of the surrounding mucosa. Peach peeling, moreover, is a rare object to be found in the trachea; in fact, we know of no similar case. The last three bring out once again the intolerance of the lower respiratory mucosa to vegetable matter. These cases, too, emphasize the importance of making a diagnosis from symptoms and indirect signs of the presence in the tracheobronchial tree of a foreign body which is not opaque to the x-ray.

Fig. 14. Anteroposterior view of large bead resting temporarily in the right main bronchus of a girl of 10. (Case 9.)

Fig. 15. Lateral view of same.

SUMMARY

Twelve cases of aspirated foreign bodies are reported. The first six were of metal and of unusual size and shape. Their removal was followed by prompt and uneventful convalescence. In the last six, on account of pulmonary suppuration, intratracheal drainage was necessary. Frequent aspirations through a soft rubber catheter in the tracheotomy tube, in our opinion, often makes the difference between life and death. In two of the cases incision of the trachea for the removal of the foreign body was necessary on account of the edema of the larynx.

144 PONCE DE LEON.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, February 1, 1937

THE PRESIDENT, DR. IRA FRANK, IN THE CHAIR

CASE REPORTS

Pulsion Esophageal Diverticulum

H. G. LAREAU

This patient was first seen on October 27, 1936, when he complained of hoarseness, pain and difficulty in swallowing, with pressure in the neck after eating. There was a bulging sac on the left side of the neck at the level of the cricoid cartilage, which increased in size after eating. On pressure by the hand, food was emptied from the sac.

A roentgenogram (barium) revealed a pushed out pouch in the upper esophagus. The anterior wall in the region of the epiglottis showed the configuration of a diverticulum. Fluoroscopic examination with barium revealed the same findings, and esophagoscopy verified the diagnosis of pulsion esophageal diverticulum.

Treatment consisted of a one-step operation on January 22, 1937. The end results were good.

DISCUSSION

DR. JOSEPH C. BECK: A one-step operation is not considered a good procedure in the majority of instances. The two-step operation, recommended by Lahey and performed by him many times, is the one generally accepted. I have had the pleasure of seeing two of the cases cured. Turning the sac upside down, emptying it and applying sutures around it so we can obliterate it easily makes a permanent cure. With the one-step operation two complications may arise, the mediastinitis if there is extensive dissection, and the other condition is dilatation at the ostium, which is always a danger.

DR. H. G. LAREAU (closing): Dr. Beck is perfectly right in stating that the two-step operation is the safest. However, there are two opinions prevalent; one school favors the one-step operation

when the pedicle is small; in my case it measured about one-half inch. In such cases the one-step is indicated. The other school favors a two-step operation when the pedicle is large and the sac extends down into the thoracic cavity. In this case the complications, such as mediastinitis, are minimized.

My patient was fed for four days postoperatively by a tube passing through the nose to the stomach, after which curds, eggnogs, etc., were fed by mouth.

There has been an uneventful recovery.

Facial Nerve Transplant

J. R. LINDSAY

CASE 1.—J. D., male, aged 47, gave a history of a simple mastoid operation on October 26, 1934. Immediately following the operation the left side of the face was paralyzed.

He was first seen in consultation on October 27, 1934. On October 28, 1934, the presenter explored the mastoid cavity and found that the facial nerve had been torn out for a distance of about 1.5 cm. from the level of the horizontal canal downwards. The facial canal was also gone in this area. The accident apparently had occurred in cleaning out extensive retrofacial cells.

The upper end of the nerve was uncovered almost to the ganglion and the lower end to the foramen, and the ends trimmed. A trough was prepared to receive the graft. A piece of intercostal nerve was removed from beneath the seventh rib and laid in the gap, with the ends in apposition with the trimmed ends of the facial nerve. The graft was covered with silver foil. About 2.5 cm. of graft were necessary to bridge the gap. Eight months later function began to appear at the angle of the mouth and gradually increased until at nine months the muscles below and above the eye had regained function. The expression in repose is now good and the patient is able to close his left eye.

Photographs and moving pictures of the case before transplantation of the graft were presented.

CASE 2. PITUITARY TUMOR EXTENDING INTO THE NASOPHARYNX

Mrs. F. C., aged 47, was seen by Dr. Lindsay in consultation on December 30, 1936. The history of the present illness was as follows: Nasal obstruction, left side, appeared sixteen months ago and increased until both sides of the nose became almost completely obstructed. Deafness in the left ear was noticed fifteen months ago

and has persisted. Sudden decrease in the vision of the right eye was noted about three months ago. Transitory occipital headaches, extending over the vertex, have occurred during the past three months.

Examination reveals a smooth mass filling the vault of the nasopharynx and extending down to the soft palate, visible also through the anterior nares. The left middle ear is filled with fluid (exudative catarrh). In the left anterior cervical region opposite the thyroid cartilage is a small, soft nodule which may be a metastasis. There is marked loss of vision in the right eye. The fundi are negative, the external ocular movements normal. General neurologic examination is negative. There are no signs of acromegaly and no symptoms of hypopituitarism.

Roentgenographic examination shows that the bone in the region of the sella turcica has been destroyed. There is gross destruction at the base of the skull. The clivus, petrous pyramids, pterygoid plates and the base of the septum have all been extensively eroded.

Biopsy from the mass in the nasopharynx reveals tumor tissue composed of a fairly homogeneous mass of cells which with hematoxylin-eosin stain show up as having dark staining nuclei and diffuse faintly staining eosinophilic cytoplasm.

Diagnosis, probable chromophobic pituitary adenoma.

Treatment: The patient has received 700 mgm. hours of radium therapy to the mass in the nasopharynx and is now receiving roentgenotherapy.

Case 3. Pituitary Tumor Invading the Nasal Septum

Three years ago I had another case with almost the exact symptomatology and the same type of tumor. The patient was a man about fifty years of age, whose only complaint was of increasing nasal obstruction of several months' duration.

Examination showed the posterior half of the septum to be dark red and so thickened as to almost obstruct the airway on both sides. The nasopharynx was clear. There was no visual disturbance. The visual fields were normal, as were the ocular movements. The patient exhibited no signs of pituitary disturbance.

Roentgenographic examination revealed practically the same degree of destruction of the pituitary fossa and base of the skull as in the first case.

Biopsy of the mass invading the septum revealed tumor tissue which was almost identical with that in the first case.

A diagnosis of chromophobic pituitary adenoma was made.

This patient was returned to his home city for treatment by radium and roentgen ray. Three weeks following his treatment he died rather suddenly from a cerebral accident, apparently hemorrhage.

(Lantern slides of roentgenograms and photomicrographs of both cases were shown.)

These last two cases were demonstrated because they represent a relatively infrequent type of tumor, and because the symptomatology in both cases was referred entirely to the nose and ear in the earlier stages.

DISCUSSION

DR. SHERMAN L. SHAPIRO: Was that the descending part of the facial nerve?

DR. J. R. LINDSAY: Yes, from the posterior portion downward, close to the foramen.

DR. FRANK J. NOVAK: I have seen tumors much smaller than this, and yet the ocular symptoms were the first that were observed. With this large mass, where the sella turcica formerly was, how do you account for the escape of the eye—how does it happen that the patient did not have very pronounced ocular symptoms?

DR. J. R. LINDSAY (closing): I do not think I can answer Dr. Novak's question, except that we must infer that the tumor was not infiltrating in character. If it was it must have involved other cranial nerves long before it reached this size. The tumor was very extensive and involved the base of the skull, yet it did not invade the cranial nerves.

Maxillary Sinus Infections

CARL H. CHRISTOPH

This case is one of a series of chronic maxillary sinus infections which we are operating upon by a modified intranasal Canfield operation at the Infirmary.

The operative technic is as follows: The incision is made anterior to and at a right angle to the inferior turbinate, and the entire mucosa of the lateral nasal wall is dissected from the bony nasal wall. A portion of the pyriform process as well as a large part of the lateral bony wall is removed, and a flap is laid into the antrum.

I believe there is a great future for the intranasal operation. In my opinion as extensive an operation can be done through the nose as by way of the canine fossa. Halle recently made the statement that there is no indication for the Caldwell-Luc procedure except in malignancy. The bleeding is usually minimum, since the blood vessels are contained in the mucosa which has been dissected away from the bony wall, and one obtains a beautiful view of the inside of the antrum. The diseased mucosa and polypi can be removed just as well as through a Caldwell-Luc incision, and I am certain that within a few years very few Caldwell-Luc operations will be performed.

DISCUSSION

DR. SHERMAN L. SHAPIRO: I would want to check the anesthesia of the teeth following such an operation.

DR. WALTER H. THEOBALD: I wish to comment on the operation Dr. Christoph reports, because some eighteen or twenty years ago the operation just described was the subject of a thesis written by me for membership in the Chicago Laryngological and Otological Society, and I think I described practically the same procedure Dr. Christoph presented. This operation approached the antrum through the pyriform crest and I reported it on about fifty patients. I cannot say I have repeated it as often since then. I have reverted more to the canine fossa operation. I titled the operation a modified Canfield operation. If we are considering priority I want to get my word in.

DR. JOSEPH C. BECK: I think Dr. Christoph should wait until these patients have been observed for some time. They often complain of having their antral discharges empty without their knowing it while they bend their head forward. This is due to the fact that the most anterolateral part of the antrum at the floor of the nose is removed. They do not feel these secretions because there is so much disturbance of the nerve supply. That is one of the great dangers in the procedure, and he may want to restrict his remarks that there will be no more Caldwell-Luc operations performed in the future.

DR. SAMUEL SALINGER: I can remember Dr. Theobald's presentation of this operation many years ago and recall that it was well received at the time. I have done the operation successfully a number of times, although I recall one instance in which I drilled for a long time before being able to enter the antrum. There are some cases in which the antral walls are very thick, due to the small size of the antrum, so that in these cases the pyriform margin represents a double thickness which is very difficult to penetrate. I would consider the

operation only where the roentgenogram reveals a large antrum with comparatively thin bony walls.

DR. CARL H. CHRISTOPH (closing): I thought I would be sat upon. We have not been doing this operation as long as Dr. Theobald and may find it is not as efficient as we now think. The advantages as I see them are, first, that we do not see the patient the day after the operation with his face badly swollen. Secondly, he does not complain of anesthesia of the teeth and, thirdly, there is very little reaction following the operation. I do not suppose it can be done in all cases. Perhaps the small antra, as mentioned by Dr. Salinger, with the thick walls are not favorable. I think the reason for failure in most cases is that the procedure was not radical enough. We dissect the lateral wall as far back as the posterior end of the inferior turbinate. We leave no ridge on the floor of the nose, and the plastic procedure is such a large one that it cannot close. In answer to Dr. Beck as to the secretion running from the nose when the patient stoops over, we have had no complaints up to the present time, perhaps because we have not observed the cases long enough.

Lymphosarcoma of Nasopharynx and Nose (Case Report)

WILLIAM A. SMILEY

Mr. J. M., aged 47, came to my office August 4, 1936, with the following history: For the past four months he had been troubled with considerable bloody discharge from the left nostril and an increasing nasal obstruction. He had consulted a physician, who had on three occasions removed "polyps" from the left nostril. About six weeks ago the right side of the nose also became obstructed. Three weeks ago he noticed a swelling in the right side of the neck, which has increased in size rapidly. It was not tender, nor was there any nasal pain or headache. No loss of appetite, weight or strength had occurred.

Examination revealed a complete obstruction of both sides of the nose by a tumor mass, which on the left side protruded from the anterior nares and also from the posterior nares, where it was continuous with the vault of the pharynx. The tumor was rather firm and bled easily. There was a right cervical lymph node about 8 cm. in diameter. Roentgenograms of the nasal sinuses showed a marked opacity of the left antrum and partial opacity of the right. Roentgenogram of the chest was negative. The differential blood count, Kahn and Wassermann reactions were negative.

A portion of the nasal tumor mass was removed and sent to the laboratory. There was very little bleeding. The diagnosis returned was "round cell lymphosarcoma. The fact that most cellular tumors in this region are transitional cell epidermoid cancer was duly considered in making the above diagnosis." The cervical gland was not disturbed.

This patient was referred to Dr. Cutler at the Michael Reese Hospital Tumor Clinic. He concurred in the diagnosis and started radiation therapy. Using the 4 gram pack, he gave a total of 162,000 mg. hours to the nose and neck, over a period of seven weeks. At the end of that time there had been a complete disappearance of the tumor mass in the nose, and the cervical gland was barely palpable. Nasopharyngeal examination was negative. There was a large perforation in the posterior part of the nasal septum.

In November the patient was given 50 mg. hours intranasal radiation on each side. At present he is free of nasal symptoms except dryness, and is in active supervision of his business. He complains of some stomach distress, which may be a forerunner of a distant involvement.

Kundrat (Über Lympho-Sarkomatosis. Wien. klin. Wchnschr., 6:211, 1893) of Vienna, in 1893, was the first to adequately describe lymphosarcoma. According to him, it is a tumor originating in a single lymph node or a group of nodes, as in the pharynx, tonsil or intestinal tract. It consists of a reticulum in which there are large numbers of lymphocytes, and after destroying the capsule of the node, it extends to other nodes.

Ewing (Radiosensitivity. Radiology, 13:313, Oct., 1929), in a series of 100 nasopharyngeal malignancies, found nine instances of lymphosarcoma.

The site of origin of nasopharyngeal lymphosarcoma is usually the lymphoid tissue of the nasopharynx. The tendency of the growth is to extend forward, involving the nasal sinuses, and in one case reported by Cutler (Lymphosarcoma, Arch. Surgery, 30:405, March, 1935), even to cause complete blindness.

Differential diagnosis must consider, in addition to squamous cell carcinoma, transitional cell carcinoma, lympho-epithelioma and aleukemic leukemia.

The clinical characteristics of this tumor are its marked radiosensitivity and its equally marked tendency to local and outlying recurrences. These, in turn, respond to radiotherapy, but repeated recurrences are the rule and the ultimate prognosis is bad.

DISCUSSION

DR. FRANK J. NOVAK: It might be interesting to know what the original polyps that were removed from the nose were, histopathologically, whether they were typical nasal polyps that most of us scorn to examine microscopically, or whether they were a true neoplasm. Were those original polyps sectioned and examined?

DR. WILLIAM A. SMILEY (closing): No, they were not. The patient consulted a general practitioner, who was a good personal friend of his, and he kindly consented to remove the polyps.

The reason the patient is not here tonight is that he has been having some stomach distress, and it seems to me there is a possibility that he may be developing some distant metastases.

Primary Cholesteatoma of the Mastoid

L. B. BERNHEIMER

(This paper appears in full on page 453 of this issue.)

DISCUSSION

DR. ALFRED LEWY: This patient of Dr. Bernheimer's was also seen by me, with the findings essentially as he described them. The cavitation in the mastoid shown on the roentgenogram was interpreted by me as a surgical cavity from the previous operation or a result of that. What the patient had was a rubber eraser deep in the canal with some bone work to remove it. I think the evidence is not clear of a primary cholesteatoma in the presence of a suppurative lesion which may have existed longer than he believed. I recently saw a cholesteatoma that had existed over many years. This young man, a musician, had had a mastoid operation several years before I saw him. He had pain in the mastoid region. Two years ago he went to a large clinic and was sent home without any surgery being done. After his money was all gone he came to the Illinois Eye and Ear Infirmary. The mastoid was inflamed but the canal was dry and there was partial atresia. I had him examined by a neurologist, who made a diagnosis of hysteria. I was not satisfied with this and kept him under observation. Within two weeks he developed an acute otitis media with discharge. The discharge had not been present for some time before that. We treated him and kept him under observation. The scar became red but there was no fluctuation. Because of the general appearance of the patient and the continued and increasing symptoms we decided to go in. We found evidence of a former

incomplete operation, because the operator came upon the sinus lying in front of the antrum and the operation had to be completed by the Küster procedure. The antrum was very deep. (Roentgenogram.) We interpreted this (indicating) as the operative cavity, which was not there. The cavity was filled with a large cholesteatoma. The operation was performed only last Friday and the patient is doing very well.

Here is a cholesteatoma due to a suppurating process which produced no symptoms except pain, in a neurotic patient, over a period of years. Now the pain is gone and the patient feels all right.

DR. GEORGE E. SHAMBAUGH, JR.: Did Dr. Bernheimer merely remove the cholesteatomatous material, or did he do a radical mastoid operation?

DR. SAMUEL SALINGER: It is generally agreed that a cholesteatoma that shows any connection with the tympanic cavity or mastoid antrum cannot be considered a true or congenital tumor. There must be a definite partition of bone separating the mass from any part of the tympanic air spaces. Most of the true cholesteatomas are found extradural near the squamoparietal or the parieto-occipital junctures and occasionally close to the dura in the petrous bone itself. In the literature which I have read during the past twenty years I can recall only two genuine, undoubted cases of primary cholesteatoma of the temporal bone on record. One case was that of a man who was brought into the hospital with an acute mastoid which was operated upon at once without the usual preoperative roentgenogram. They did a radical mastoid operation and two weeks later took a picture to see how it was getting along, and to their astonishment found a smooth cavity posteriorly that seemed to be overlying the mastoid. A second operation disclosed a true cholesteatoma which was separated from the cavity operated upon by a dense lamella of bone.

The second case was in the petrous bone, demonstrated by the roentgen ray, which was operated upon by withdrawing spinal fluid and doing a radical mastoid operation exposing the middle fossa and superior surface of the petrous bone, when the searcher entered a cavity just beyond the labyrinth, a cavity from which large amounts of cholesteatomatous material and part of the matrix were removed. While in Dr. Bernheimer's case there is no definite history of infection, nevertheless the history of trauma and the involvement of the peri-antral region would lead one to believe that the resultant tumor formation was more than likely due to secondary invasion of epithelial tissue rather than congenital inclusion.

It has been shown by Wittmaack and others that extreme retraction of Shrapnell's membrane may cause an invagination into the attic and antrum leading to the formation of cholesteatoma in spite of definite absence of history of any previous suppuration.

Dr. Walter H. Theobald: I believe the question of differentiating between primary and secondary cholesteatoma is debatable, but the younger the child, the more likely one is to find primary cholesteatoma. By a peculiar combination of circumstances I saw a case last week. This patient is now about 24 years old and was first seen by Dr. Pierce in October, 1914. He was then about three years old and had had a discharging ear for three months without any known cause. He had no pain nor other symptoms referable to otitis media. He had been under the care of pediatricians, and suddenly a creamy discharge was noted coming from his ear. Polypi were removed and subsequently a radical mastoid operation was advised. At the time of operation a diagnosis of primary cholesteatoma was made. Cholesteatoma was found invading the mastoid, although there had been no evidence of this material invading the middle ear. A dry ear was accomplished subsequently, after a very trying time. This young child required many months of after treatment. The hearing was totally absent then and has been ever since. When I saw the patient a week ago the ear was again moist, but there was no evidence of cholesteatoma. This is one of those unfortunate mastoids in which moisture in the ear recurs.

Dr. L. B. Bernheimer (closing): Answering Dr. Shambaugh, a simple antrotomy was done. The antral lining was found to be clean. Inasmuch as the drum membrane was normal, no indication for a radical operation was seen.

The one point that speaks against primary cholesteatoma is that a postauricular incision had been made to remove a foreign body. It is possible that epithelial tissue was displaced into the mastoid at that time. However, inasmuch as no evidences of bone work were encountered at the time of the antrotomy, it is difficult to understand how such displacement could have taken place.

The cholesteatoma followed Dr. Salinger's rule for primary cholesteatoma, as it was walled off from both antrum and middle ear by trabeculæ of normal bone.

Inasmuch as there was no history of a previous otitis, and inasmuch as no evidences of infection were encountered in the mastoid, this particular case was in no way parallel to the cholesteatoma described by Dr. Lewy.

The Human Tooth as a Recorder of Biologic Processes With Special Reference to the Neonatal Ring

ISAAC SCHOUR

(Author's Abstract)

Experimental and clinical evidence has established the fact that the growing tooth is a very delicate and accurate indicator of biologic processes. Erdheim's analogy that the dentin of the incisor of the rat acts like the drum of a kymograph in its response to disturbances in calcium metabolism is applicable to enamel as well as dentin, to various metabolic changes and to the growing human teeth.

Injections of sodium fluoride or alizarine produce a permanent mark or ring in the portion of the tooth that is calcifying at the time of the injections. This method makes possible the accurate measurements of the rate of growth of enamel and dentin. In the rat incisor there is a 16 μ daily incremental rhythm. In the human tooth, the rate of growth is approximately one-fourth as fast.

In the attempt to analyze the rings that were produced experimentally a number of other rings have been discovered, each of which is constant and characteristic in position and is therefore produced by a common cause. One of these constant rings is the neonatal ring which is found in every human deciduous tooth and which constitutes a permanent biologic landmark. The neonatal ring mirrors the biologic changes in the dental tissues of the newborn child that may result from metabolic influences that are incident to a change from intra-uterine to extra-uterine existence.

The presence of other constant physiologic rings, as well as the occurrence of rings induced by known diseases, indicate the value of tooth-ring analysis as an aid in the diagnosis of health and disease.

DISCUSSION

DR. WALTER H. THEOBALD: This has been a very dramatic and fantastic story, and we are very grateful to Dr. Schour for his splendid presentation. Dr. Kronfeld, whom Dr. Schour mentioned, of the Dental College of the Loyola University, has been much interested in this work, and I have invited him here this evening to discuss Dr. Schour's presentation, as well as to provide a link between this work and the sinuses. Dr. Kronfeld has made an exhaustive study on the maxillary sinuses in relation to the teeth, and I should like to have the Chair call on Dr. Kronfeld.

DR. RUDOLPH KRONFELD: I certainly appreciate Dr. Theobald's invitation to come down here tonight to listen to Dr. Schour's report of his work. We have known of his investigations for many years and have followed them with great attention and appreciation, for we have felt, as he pointed out, that science, especially morphology, has reached a point where it is no longer sufficient simply to say that a structure or tissue grows, but we want to know how much it grows in a certain length of time, and then proceed to an understanding of the anomalies in pathologic events. I hope you enjoyed Dr. Schour's presentation as much as I did.

Dr. Theobald asked me to bring along some material on the pathology of the teeth and the maxillary sinus. Our work has been along a little different line. What we have done is to examine about eighty human jaw specimens, ranging in age from newborn to eighty years, all of which were roentgen-rayed, decalcified and studied in sections. From this material we can tell, for instance, how far the teeth are developed in the jaws of a child of one, two, three or four years of age. We can study the normal and abnormal jaw tissues; in the latter we find many cysts and other pathologic conditions, and we can then compare what we find microscopically with the clinical picture.

In sectioning the upper jaws we have always paid attention to the maxillary sinuses and their relation to the root ends in normal and abnormal conditions, as well as the relation of the nose to the incisor teeth. I shall show you, by means of lantern slides, a few things that I believe will be of interest to you.

(Presentation of lantern slides showing the growth of the maxillary sinus in children, the relationship between the root ends of the upper posterior teeth and the floor of the antrum, and the effect upon the antrum of granuloma and cysts originating from the upper teeth.)

DR. WALTER H. THEOBALD: I should like to ask Dr. Schour if he can give us a word with reference to his study upon the failure of enamel development that we see sometimes on the central incisors.

DR. ALFRED LEWY: What dilution of sodium fluoride was used?

DR. H. G. LAREAU: With reference to the line markings on teeth, as described by Dr. Schour, I would like to ask him if he could deduce the possibility of these markings being used in a practical sense by horse buyers and traders.

DR. I. S. SCHOUR (closing): I want to state that these illustrations and models which we have shown you were made principally by Dr. Spence, who assisted me in presenting this material.

In regard to the question of enamel hypoplasia, no doubt many of you have noted definite pits on the central incisors and the cuspids, usually not on the laterals. These are disturbances that occurred when the enamel was calcified. By checking up the amount of enamel and dentin on a tooth we can tell definitely when these marks were formed in the early years of the life of the individual. We have further evidence in the fact that the centrals and cuspids will show this but not the laterals. Dr. Kronfeld has found that the laterals start forming much later than the others, usually at about the second year. So we know something happened before that time. The probability is that we are dealing with individuals who had tetanic convulsions about that time. These convulsions are more likely to occur at about the last of the first year. Sometimes individuals will give a history of convulsions at that time. The explanation is that the first year of life probably marks the most rapid period of growth in the life of the child. When the individual grows rapidly the disturbances of calcium metabolism are more effective. When there is slower growth these changes may not be registered.

Answering Dr. Lewy, we used about 2 per cent sodium fluoride and 1 cc. or 1.5 cc. was sufficient. The injections were given intravenously, which made it very simple. There were no markings of any sort. Careful temperature records were kept, careful medical attention was given the child and the medical history showed no untoward effects of the injections of sodium fluoride except in the teeth. The child died of confluent pneumonia and the complications of the hydrocephalic condition.

With relation to marks on teeth, it is quite evident that if the neonatal life is sufficient to produce marks there must be other experiences that are recorded in the teeth. We are now studying changes in rats and are finding weaning rings. When we put animals on one diet and then change to another we do find differences. When the mother stops giving the child milk there is a change in diet, and this sudden change produces a definite ring in the teeth. This ring not only acts as a boundary but it also indicates changes in calcification. It is logical to expect to find a puberty ring. We are almost confident that we will find a puberty mark, but we will have to look for it in the third molar. It is possible that we shall be able to tell whether a third molar comes from a female or male person, for puberty occurs earlier in the female.

With relation to telling the age of the horse by examining the teeth, my impression is that this depends, not on the rings, but on the degree of abrasion which progresses with age.

DR. RUDOLF KRONFELD: There is a difference in the structure of the teeth of man and the horse. In man there is only one layer of enamel, whereas in the horse the enamel is folded in, so that there is a funnel shaped layer of enamel inside the tooth. As the animal grows older, the outer layer of enamel stays about the same width, but the inner layer grows narrower and narrower, as the teeth wear down. Thus, from the difference in the size of these two enamel rings on the abraded surface, it is possible to tell how old a horse is.

DR. I. S. SCHOUR: Another instance in which the age of an animal can be told happened in Switzerland. A man was arrested for hunting a stag, with the accusation of breaking the law, for it is against the law to hunt a stag that is less than six months of age. This man brought a dentist for a witness who, by examination of the teeth of the stag, was able to tell the exact age of the animal. By counting the number of rings in the tooth, he was able to tell the exact age of this stag because the annual ring is formed in the secondary dentin.

Books Received

Memoranda of Toxicology.

Max Trumper, B.S., A.M., Ph.D., Consulting Clinical Chemist and Toxicologist. Cloth. 18mo of 304 pages. Third Edition. Philadelphia: P. Blakiston's Son & Co., Inc., 1937. Price, $2.00.

This little book, hardly a handful, is a most convenient manual of the behavior, or better the misbehavior, of drugs. The material is tersely written and practical. Along with the short description of symptoms and antidotes, there is often room for a few paragraphs on the derivation of drugs and some of the colorful background with which poisons are invested.

Something to carry in the grip.

Physical Therapeutic Methods in Otolaryngology.

Abraham R. Hollender, M.D., F.A.C.S., Associate in Laryngology, Rhinology and Otology, University of Illinois College of Medicine; Fellow of the American Academy of Ophthalmology and Otolaryngology. Cloth. Octavo of 442 pages with 189 illustrations. St. Louis: The C. V. Mosby Company, 1937. Price, $5.00.

This is a complete textbook on physical and mechanical methods in otolaryngology. It deals not only with various electrical appliances but with other physical agents as well. The author is to be commended for his conservative approach and careful evaluation of the various methods. Each chapter is followed by a comprehensive list of references. The histopathology of each subject is adequately dealt with.

Diseases of the Nose, Throat and Ear.

I. Simson Hall, M.B., Ch.B., F.R.C.P.E., F.R.C.S.E., Surgeon to the Royal Infirmary, Edinburgh, Lecturer in Diseases of Nose, Throat and Ear, University of Edinburgh Cloth. Crown 8vo. of 440 pages with 55 text Illustrations and colored frontispiece. Baltimore: William Wood & Company, 1937. Price, $4.00 net.

This is a convenient manual in small format, for the student and the general practitioner. It is quite complete in the material dealt with, but the size of the volume precludes any very detailed discussions of it. It is recommended as a handbook for those not specializing in otolaryngology rather than as a manual for the student of the specialty, who will demand a more detailed text.

Plastic Surgery of the Nose.

J. Eastman Sheehan, M.D., F.A.C.S. Cloth. Octavo of 186 pages with 131 text illustrations, including seven in color and fourteen full-page plates consisting of 104 photographs. Second Edition, entirely rewritten. New York: Paul B. Hoeber, Inc., 1936. Price, $9.00 net.

This is a revision of Plastic Surgery of the Nose, published in 1925. The particular revision which characterizes this book is the recognition of the interrelationship of the various deviations from the normal. The author feels that corrective operations must take into account that where there is one deviation from the ideal anatomy, there are usually others. Unless all of these are corrected in harmony, the nasal disfigurement persists.

Special attention is called to the behavior and utility of skin grafts, the result of years of experience and new conceptions of the possibilities in readjustment and reconstruction of the nasal framework.

The material is divided into six sections: I. Anatomy and Function; II. Typical and Distinctive Operations; III. Classified Corrections at the Three Nasal Levels; IV. Losses; V. Skin Grafts; VI. Precaution, Preparation and Postoperative Care.

Illustrations and typography are of the best.

Abstracts of Current Articles

NOSE

Treatment of Depressed Fracture of the Malar Bone.

Watkins, A. B. K. Brit. Med. J., p. 326 (Feb. 13), 1937.

A fracture which is becoming common is that in which the malar bone is pushed into the antrum. The orbital, antral and zygomatic attachments of the malar bone are fractured. The malar bone being strong is rarely comminuted, but not so the lateral wall of the orbit and the infra-orbital margin of the maxilla. If the case is seen soon after the accident the diagnosis is easily made, but even if seen after several hours the depression of the malar bone is rarely masked by the superficial swelling. When there is much swelling an alteration in the shape of the lower edge of the orbit is generally such as to make the presence of this fracture highly probable. The diagnosis in difficult cases is made by x-rays, and especially by one taken vertically of the side of the head from below upwards, when the prominence of the zygomatic process on the affected side will be absent. Often the fracture causes loss of sensation over the area of distribution of the infra-orbital nerve. As a rule, the antral cavity is filled with blood clot.

Treatment by the usual methods is often unsatisfactory. A common procedure is to make an incision under the lip similar to that in a Caldwell-Luc antrostomy and to open the antrum and to elevate the malar bone. The reduction of the fragment is easy by this method, but no facilities are provided for retaining the bone in the correct position, and no allowance is made for drainage of the antrum. Silverman uses tenaculum forceps, which seize the malar bone through the substance of the cheek, but the amount of superficial swelling or degree of displacement into the antrum would often preclude this method. The method of Gillies is to pass an elevator through a small incision above the hair line in the temporal region. This is maneuvered down on the surface of the temporal muscle under the root of the zygomatic process, which is then levered outwards. This method is often an excellent one, but it does not always succeed in retaining the bone in the correct position.

The author's method of reducing the fracture is by working through an intranasal antrostomy opening. The operation is performed by a vertical incision in the nasal fossa from immediately above and in front of the anterior extremity of the inferior turbinate bone to

well on to the floor of the nose. This incision is carried as deep as the antero-medial border of the maxilla, and the bony opening is carried backwards into the inferior meatus. An elevator is then inserted and the body of the malar bone is elevated into position. Usually the displacement has no tendency to recur, but if it does this is easily controlled by packing the antrum. This method provides an easy and certain means of reduction and retention of the fragment in position. It usually makes the patients quite comfortable as soon as the drainage of the antrum is provided.

GOLDSMITH-MCLEOD.

Spreading Osteomyelitis of the Frontal Bone Secondary to Disease of the Frontal Sinus, With a Preliminary Report as to Bacteriology and Specific Treatment.

Williams, Henry L., and Heilman, Fordyce R. (Rochester, Minn.) Arch. Otolaryng., 25:196. (Feb.), 1937.

The finding of the identical organism, an anaerobic streptococcus, in two cases of osteomyelitis of the frontal bone, together with the apparently unusually favorable results obtained by specific therapy with an autogenous antivirus, is suggestive that the organism causative of this lesion may have been isolated. This hope is intensified by the fact that previously there has not been any adequate explanation as to why in an occasional case of frontal sinusitis this lesion should develop, either spontaneously or after operation, when the bone is equally exposed to infection after any type of infection of a sinus or intervention which involves it. The conjecture that the disease is of staphylococcic origin has not seemed satisfactory, because staphylococci frequently are present in sinuses which have been operated on without producing this type of pathologic change. We present our findings with the hope that they will be confirmed in further investigations by ourselves and by others. Treatment with the antivirus clinically seemed to have a favorable effect in these two cases.

We feel that delaying radical operative procedure as long as it is consistent with good surgical judgment is best. We believe that this allows the natural resistance of the patient to be built up against the infection, especially as the natural resistance seems to be feeble, and built up slowly in this type of case. The decision as to the optimal time for surgical intervention will be influenced considerably by the rapidity with which the inflammation spreads. In fulminating cases in which meningitis and sepsis often appear in from twelve to twenty-four hours after the first appearance of symptoms, we believe that radical surgical operation tends to hasten the spread of the disease and that only enough should be done to relieve the pressure and drain the pus that is within the frontal sinus. If the fulminating stage

subsides the surgeon should be guided by developments. We feel that the inflammatory change in the dura is the best guide to how far removal of bone should extend. It seems logical to treat the manifestations of the disease in the sinuses at the time when the diseased frontal bone is being removed. In consideration of the fact that Furstenberg and Mosher have demonstrated that propagation of the disease is by thrombosis of the dural veins which communicate with the dural sinuses and the intradural veins, it would seem best to eradicate the primary disease in the bone before attempting to treat complications, such as suppurative encephalitis or thrombosis of the dural sinuses. TOBEY.

Sclerosing Submucosal Injection of the Turbinates.

Hutchinson, C. A. Brit. Med. J., p. 72 (Jan. 2), 1937.

The author believes that, in the treatment of conditions such as early hypertrophic rhinitis, nonspecific hay fever and nasal asthma, there is far too great a tendency to have recourse to the thermocautery. The risks of cauterization are the production of adhesions and excessive damage to the ciliated epithelium. Adherence of nasal secretion, with inspissation and crusting, results from the loss of the ciliary stream. In the course of time ulceration occurs, and the condition drifts on into a state of atrophic rhinitis. The writer has obtained very satisfactory results from a sclerosing submucosal injection of the turbinates, which aims at producing scarring in the erectile tissue of the subepithelial layer of the mucosa and in the submucosal space, very little damage being done to the ciliated epithelium. Carbolic in paraffin, 30 per cent, is used for the injection and the technic is given in full, as strict attention to detail is of the utmost importance. The shrinkage produced is excellent, and close adherence of the mucosa to the underlying bone is secured. The procedure rarely has to be repeated, and when it does is still comparatively harmless.
GOLDSMITH-MCLEOD.

The Maxillo-frontal Canal (Le canal maxillo-frontal).

Mangabeira-Albernaz, P. (Campinas). Rev. de Lar., Otol., Rhin., 58:77 (Jan.), 1937.

Although ignored by most authorities, a canal leading directly from the maxillary antrum into the frontal sinus was found in two of our sixty-eight cases and demonstrated at operation by probe and radiographs. The canal runs down from the most anterior internal portion of the frontal floor, in front of and external to the nasofrontal duct. Cadaver studies by Vilar Fiol, who first described this canal in 1928, suggest that it occurs in rudimentary form in almost 20 per cent of 140 cavities. Laterally flattened, the canal lies behind the

lacrimonasal duct, and is seen above the normal ostium in the postero-internal angle of the maxillary roof. In other cases the author has found a short canal in this region, running up into the hiatus but not communicating with the frontal.

FENTON.

Ossifying Fibroma of the Maxillary Sinus: Report of a Case Successfully Treated With Radiation.

Arons, Isidore, M.D., D.M.R.E. Am. J. Cancer, Vol. 24, No. 3 (March), 1937.

Photographs, radiographs and microphotographs are shown and description given of a tumor noticed in the maxillary sinus of a white boy, 11 years old. Swelling of the right side of the face occurred July, 1927. Two teeth were extracted in September, 1927. Histologic diagnosis of a piece of tissue on a root of one tooth was osteogenic sarcoma. Radiographs revealed a circumscribed area of bone density filling the right maxillary sinus. In October, 6,000 mg. hours of radium were given through a wax mold 3 cm. thick, 0.5 platinum and 1.0 mm. brass filtration applied to cheek for forty-eight hours. From November 26 to December 14, eight injections of Coley's toxins were given, beginning with 1 minim and increasing to 8 minims. Two months following radium application, twelve x-ray treatments to three fields, 200 "r" each treatment, were given. Nine doses Coley's toxin commencing with 2 minims and increasing to 16 minims were given simultaneously.

Ten years later patient showed no evidence of recurrence or metastasis.

The heavy radiation dose was administered on account of the diagnosis of osteogenic sarcoma. At a later date Dr. Geschickter made a diagnosis of ossifying fibroma. This case is interesting and instructive in that the result indicates that radiation may successfully irradiate this benign type of neoplasm instead of the usually recognized procedure of surgical extirpation.

JORSTAD.

PHARYNX

Diagnosis of Malignant Disease of the Pharynx.

Pilcher, Robin. Brit. Med. J., p. 13 (Jan. 2), 1937.

The chance of cure which lateral pharyngotomy holds out might have been expected to stimulate interest in the early diagnosis of this common form of malignant disease. It is a fact, however, that recognition of these growths in their early stages, upon which any successful application of surgical excision depends, is still a rare event.

The early symptoms of this disease are trivial in nature, and although superficially resembling trivial symptoms of less significance they have certain peculiarities which should always arouse suspicion of malignancy. The lack of familiarity with these early symptoms is doubtless largely due to the overwhelming nature of the major symptoms which always supervene sooner or later. The patient with severe dysphagia, urgent dyspnea or continual pain is unlikely to dwell on the minor disturbances which preceded them, and in the presence of an obvious case of malignancy there is little temptation to the busy practitioner to probe into the past history. In the majority of cases, when symptoms first appear, some abnormality can be seen by indirect laryngoscopy; it is in a wider use of this simple method of examination that there lies most hope for an improvement in diagnosis.

The author discusses the disease under two main groups: (1) Epilaryngeal group, and (2) postcricoid carcinoma. In the epilaryngeal group the sites at which the neoplasm usually starts are: (1) The epiglottis, (2) the aryepiglottic fold, (3) the lateral wall, and (4) the pyriform sinus. In this group the commonest early symptom is a persistent abnormal sensation in the throat which is variously described; it may be a sore throat, a tickle, a feeling of a lump, or a little pain on swallowing. Whichever of these it may be, there are two features that should arouse suspicion of malignancy. The first is that the patient is able to locate the sensation to a definite part of the throat and the second is its persistence. Another symptom that may be present early is some interference with the voice. The presence of the tumor may alter the resonance of the voice, or it may infiltrate the arytenoids and limit the movements of the cords.

In the history of the typical case of post-cricoid carcinoma there are two features which, if their significance were realized, would always arouse suspicion of malignant disease, but which unfortunately more often have the reverse effect of allaying such suspicion. The first is that the patient says that she has had for many years, sometimes all her life, some slight difficulty with deglutition. This all too often makes one suspect the patient of hysteria, whereas it is a most important indication of the possibility of postcricoid cancer. The second is that, when other symptoms are added, they are at first intermittent. Perhaps the patient has a sudden difficulty during one meal with a fit of choking or a sensation of a bolus sticking in the throat, and then no more trouble for a week or two. The fact that the dysphagia is a single incident, or appears as a few such incidents separated by intervals of freedom, is very likely to lead to its dismissal as something trivial. So important does the writer regard the com-

bination of symptoms that he considers a single serious attack of dysphagia in a patient with a long history of slight difficulty to be an indication for a thorough examination, if necessary including esophagoscopy.

Common to both groups, and in this series more frequent in the epilaryngeal, is the occurrence of glandular enlargement as the first symptom. Any patient at or past middle age who develops enlarged glands in the neck should be suspected of malignant disease. The teeth and tonsils seldom give rise to such enlargements for the first time in the second half of life. There can be no doubt that sepsis plays an important part in the evolution of a pharyngeal cancer, and its aggravating effect on the symptoms is such that the chances of early diagnosis are much greater when it is present than when the mouth is clean. Moreover, so dramatic may be the improvement after the elimination that no case can be dismissed as inoperable on the local signs in the pharynx before the teeth have been extracted. It is perhaps unfortunate that the majority of women who develop postcricoid cancer are edentulous. It may well be for this reason that, when symptoms first occur in this type, the growth is often surprisingly large. The only certain way to detect an early carcinoma in this group is by esophagoscopy, and it requires courage to insist on this when the patient's symptoms are still of a trivial nature.

The author gives the results of the application of surgery in 100 cases of carcinoma of the pharynx. The mortality of the treatment is high, partly on account of the advanced state of the disease in many cases but also on account of the severity of the operation. Seven of the hundred patients are alive as the result of surgical treatment alone, the longest interval since operation being ten years, the shortest eighteen months. Other cases in this hundred have been relieved by radiotherapy, but there are as yet no long standing survivals as a result of this treatment. These few successes show that these forms of malignant disease are curable by surgery. It has yet to be shown that any other method gives better results but, whatever method of treatment is favored, it will obviously have more chance of success if diagnosis is made early.

GOLDSMITH-MCLEOD.

Abscess of the Sublingual Fossa (L'abscès de la loge de la glande sublinguale).

Citelli, S. (Catania). O. R. L., *Int.*, 20:781 (Dec.), 1936.

Professor Citelli draws attention to unilateral swellings beneath the tongue, as distinguished from glossitis proper, Ludwig's angina or salivary lithiasis. Local swelling and pain are relieved by incision

outside the sublingual fold close to the jaw, to the depth of 2 cm., reaching the areolar tissue surrounding the sublingual gland, which may become infected from bad teeth or from acute mouth inflammations.

FENTON.

Some Tumors and Ulcers of the Palate and Fauces.

Howarth, Walter (London). J. Laryng. and Otol., 52:1 (Jan.), 1937.

This is a report of the author's personal experience in tumors of the palate and pharynx over a period of twenty-five years.

Bilateral tumors are not so uncommon as is often supposed but are nevertheless relatively rare and of great variety. This region lends itself readily to the Kohnheim theory of tumor origin. Varied histologic appearances are met with and so-called "mixed tumors" preponderate. Hemangioma and hemangiofibroma are rare. The author has seen only one fatty tumor and one adenoma. The epitheliomata (61 cases) are described in some detail.

Of especial value are the color plates illustrating the various types of tumors and ulcers.

Diphtheritic Paralyses of the Soft Palate (Das paralysias diphtericas velo-palatinas).

Müller-dos-Reis, W. (Rio de Janeiro). A Folha Medico, 25 (Sept.), 1936.

Considering that postdiphtheritic palatine paralyses indicate that antitoxin was used late and insufficiently, Müller-dos-Reis has found foreign protein therapy effective in securing recovery. Fixed toxin in the nerves affected can be neutralized by strong protein reactions to which he attributes an intensification of the reticulo-endothelial defensive system. Five cases are reported.

FENTON.

LARYNX

The Problem of Early Laryngeal Tuberculosis.

Blegvad, N. Rh. (Copenhagen). J. Laryng. and Otol., 52:153 (Mar.), 1937.

These observations are based upon a series of cases treated in Copenhagen between 1916 and 1934. There were 1,773 cases of laryngeal tuberculosis. Seventy-four per cent died and 26 per cent are living and well.

The treatment was by means of arc or quartz lamp. In certain cases galvanocautery, amputation of the epiglottis, excision of the dis-

eased parts, alcohol injection or resection of the superior laryngeal nerve were also resorted to.

The author cites Thomson's list of symptoms suggesting laryngeal tuberculosis: Congestion limited to one part of the larynx and lasting some time; thickness or congestion of one arytenoid, of one aryepiglottic fold, or of one side of the epiglottis—all unsymmetrical affections. The author adds: (1) Isolated redness of a vocal cord; (2) swelling and redness of the vocal process; (3) prolapse of the ventricle of Morgagni; (4) swelling of the lower surface of the vocal cords; (5) swelling of the mucous membrane in the interarytenoid region, and (6) a red cushion beneath the commissure.

Upper Dysphagia.

Paterson, D. R. (Cardiff). J. Laryng. and Otol., 52:75 (Feb.), 1937.

This type of dysphagia is primarily related to a change in the mucosa of the upper esophagus, the nature of which is not quite clear. Its association with secondary anemia of obscure origin in women of reproductive age, and often accompanied by atrophic changes in the mouth, pharynx, nares, giving the patient a contracted appearance, suggests a wider basis and an underlying possible constitutional factor. The question is raised whether this condition predisposes to malignant disease and whether effective antecedent treatment of the anemia with iron would not prevent it.

Abnormal Forms of Tuberculosis Simulating Cancer of the Larynx and Their Converse.

Hautant, A. (Paris). J. Laryng. and Otol., 52:65 (Feb.), 1937.

Laryngeal tuberculosis can take the aspect of a warty, subglottic and unilateral lesion, or sometimes it resembles a ventricular tumor. In both instances the clinical aspect is that of an intralaryngeal epithelioma; a very careful examination is necessary to avoid an error.

A radiograph of the larynx can be a valuable aid in the diagnosis of cancer.

In doubtful cases several biopsies should be carried out from different parts of the laryngeal lesion.

Deep roentgenotherapy, even as a test treatment, should be resorted to only with great caution.

Even in cases in which a diagnosis of epithelioma seems obvious, a pulmonary examination should always be made and taken into consideration.

ESOPHAGUS, TRACHEA AND BRONCHUS

Direct Bronchoscopic Investigation in Hemoptysis Without Physical or Radiological Manifestations.

McGibbon, John E. G., and Baker-Bates, E. T., Brit. Med. J., p. 109 (Jan. 16), 1937.

In certain cases of hemoptysis there is not infrequently a lack of signs on complete clinical examination and direct or contrast radiologic study, so that it is impossible by these means to ascertain the cause or source of the bleeding. In a large proportion of cases hemoptysis is caused by pulmonary tuberculosis, and it is not surprising, therefore, to note in the older literature the great significance accorded to blood spitting in the diagnosis of the disease. Commonly the majority of patients who spat blood were regarded as cases of pulmonary tuberculosis, and many correctly so diagnosed succumbed to the disease; the remainder either died as the result of an unrecognized causative lesion or lived an invidious existence under the stigma of "consumption." Vinson concluded from his observations that pulmonary tuberculosis was regarded as the usual cause of hemoptysis merely because of the frequent and widespread prevalence of the infection, whereas the actual relative incidence of hemorrhage was greater in the other less commonly recognized pulmonary lesions. Wurtzen recorded in a large series of cases admitted into a sanatorium that in approximately one out of every three cases the cause of the hemoptysis had been misinterpreted. Today the bronchoscope is very slowly being recognized as an instrument of precision for the diagnosis and treatment of bronchial and pulmonary lesions and not merely as a mechanical means for the extraction of foreign bodies from the bronchial tree.

The writer reports six cases suffering from recurrent hemoptysis who exhibited no physical or radiological signs of pulmonary disease. In these six cases the causative lesion was found by bronchoscopic examination to be one of the following: tracheal varix, chronic nonspecific granuloma, simple new growth or malignant neoplasm. Bronchoscopy affords the only possible means of making an early and positive diagnosis in cases of bronchial neoplasm. From the patient's point of view all bronchial neoplasms are "malignant," whether histologically so or not, in that eventually they will give rise to complications which, if left untreated, usually prove fatal. Furthermore, if any successful treatment by surgery, radium or x-rays can be evolved for bronchial carcinoma its success will depend on the earliest possible diagnosis.

GOLDSMITH-MCLEOD.

Dysphagia With Anemia in a Male.

Watson-Williams, E. Brit. Med. J., p. 1140 (Dec. 5), 1936.

The association of dysphagia with anemia was noted over sixty years ago; more recently a third symptom, stomatitis and glossitis, has been added to complete the syndrome usually known as the "Plummer-Vinson syndrome." The condition is not of great rarity, but most observers agree in describing it as a disorder confined to women. The writer reports an apparently typical case in a man, aged 71 years. He complained of difficulty in swallowing during two months with no appreciable loss of weight. He was very pale and had glossitis, with loss of papillæ except in the center of the tongue. There was a linear scar from each angle of the mouth, and when questioned said that the corners of his mouth had been sore for several years. Esophagoscopy showed at the upper end of the esophagus a thin white demilune web round the posterior aspect which reduced the lumen to 8 mm. across. It yielded readily to slight pressure from the endoscope, with just a trace of bleeding. Below, the esophagus was normal except for pallor of the mucosa. Immediately following the instrumentation swallowing became normal.

The etiology of this condition is in dispute. Paterson and Brown Kelly regard the dysphagia as primary, with anemia due to interference with nutrition. On the other hand, Johnson considers the changes in the mucosa secondary to the anemia. The writer agrees with the latter view and in his experience the formation of a thin web at the esophageal entrance, instead of the spasm, is the more than usual finding.

In the treatment the administration of iron in large doses is essential. Inorganic preparations are to be preferred; the daily dose of iron and ammonium citrate is 60 to 120 grains for eight weeks or more. It may be necessary to repeat treatment during several years. Where spasm or webbing is present, treatment should begin with dilatation of the esophagus; this rarely needs to be repeated. The prognosis is good, although it is said that later malignant changes in the esophagus are relatively frequent in these patients.

GOLDSMITH-MCLEOD.

Bronchoscopy and Its Clinical Application, With a Short Account of Bronchial Catheterization.

Frenckner, Paul, and Björkman, Stig (Stockholm). J. Laryng. and Otol., 52:233 (April), 1937.

Bronchospirometry is the volumetric determination of the gaseous exchange of the respiratory air of each lung separately, brought about by the absolute isolation of the air of each lung by means of a

bronchoscope or catheter-like instrument. The method of doing this is described in great detail, which does not lend itself to effective abstraction.

The authors mention two facts which have become apparent from their investigations. The output of carbon dioxide from one lung has no connection with its intake of oxygen but with its ventilation. When the volume of tidal air is great and the oxygen intake is small, the output of carbon dioxide follows the ventilation. Therefore there may be a considerable difference between the respiratory quotients in the right and left lungs.

EAR

Structure and Function of the Membrana Tectoria (Über Bau und Funktion der Membrana tectoria).

Wittmaack, K. (Hamburg). Acta Otolar., 24:397 (Nov.-Dec.), 1936.

Calling attention to the necessity of intravital examination of cochlear end-organs, Wittmaack gives out his present opinions regarding the organ of Corti. This organ and its membrane form a common tissue complex closed against the endolymphatic spaces and indicating a definite condition of turgescence which depends upon pressure relations within the internal ear. Adequate stimulation for the cochlear nerves consists in the periodic oscillations of pressure communicated to the endolymph by movements of the footplate of the stapes. These oscillations are transmitted to the sensory nerve fibers not by the effects produced upon sensory cells but much more directly, through the turgescence effect of the membrane of Corti and of the whole tissue complex of the corresponding organ. Wittmaack promises further confirmation of this application of the laws of hydrodynamics to hearing within the current year.

FENTON.

Exostoses of the External Auditory Meatus, a Technic for Their Surgical Removal.

Miller, Arthur. Brit. Med. J., p. 263 (Feb. 6), 1937.

Exostoses of the auditory meatus are due either to partial hyperplasia during the stages of development and ossification, or to chronic suppuration of the middle ear, which causes a periostitis, with subsequent new bone formation. Exostoses are often bilateral and multiple, usually sessile, and of slow growth; they never become malignant and are said to occur more often in men than in women. It is generally accepted that exostoses should not be interfered with unless

they cause untoward symptoms. The following are the indications for their surgical removal: (1) Deafness and tinnitus; (2) incarceration of wax which cannot be removed by syringing; (3) obstruction to the outflow of pus from concurrent middle ear disease in order to establish free drainage and an opportunity for treatment of the middle ear cavity; (4) dermatitis with secretion, and (5) pressure symptoms with neuralgic pain.

Surgical removal of exostoses of the meatus is an operation which might present great difficulties and is not devoid of danger to the patient because the great hardness of the bone causes the chisel to drive on when the last fragment is divided, and it would be easy to damage the facial nerve and even the cochlea. For their removal the writer uses a burr propelled by an electric dental drill instead of the chisel and mallet. Owing to the rapidity of the motion of this machine, very little pressure is necessary, and the burr is therefore not likely to slip and cause any injury to the surrounding structures. As the bony prominence is reduced very gradually there is no danger of splitting or fissuring the bone. Another advantage is that very little bleeding takes place, as the heat produces a quick coagulation of the blood. In future the writer intends to tackle the exostoses through the external auditory meatus, provided they are not too near the drum.

GOLDSMITH-MCLEOD.

The Vestibular Apparatus and the Vegetative Nervous System (L'appareil vestibulaire et le système nerveux végétatif).

Kotyza, F. (Prague). Acta Otolar., 25:51 (Jan.-Feb.), 1937.

Making tracings of the oculocardiac and solar reflexes in twenty patients, nineteen with normal hearing, doing first the Kobrak irrigation and an hour later rotation with head 30° forward, the author finds marked effects during the nystagmus, especially increased parasympathetic tonus on rotation. Minimal stimulation, from the Kobrak tests, decreases vegetative tonus; the especial weakening of sympathetic responses may be explained by the effects of warm water irrigations upon vagus fibers reaching the external auditory meatus. Sudden stimulation as called forth by rotation brings stronger effects, particularly on the parasympathetic responses.

FENTON.

The Renaissance of Otology: Joseph Toynbee and His Contemporaries.

Guthrie, Douglas (Edinburgh). J. Laryng. and Otol., 52:163 (Mar.), 1937.

This article will be of unusual interest to lovers of the historical phase of otology. It is liberally illustrated.

MISCELLANEOUS

Osteomyelitis of the Skull of Otitic and Paranasal Sinus Origin.

Behrens, Herbert C. (Whittier, Calif.). Arch. Otolaryng., 25:272 (Mar.), 1937.

The conclusions drawn from the literature and the cases reported are as follows:

1. In the majority of cases osteomyelitis of the skull follows surgical intervention and is also more serious from a prognostic standpoint than that which arises spontaneously.

2. It is suggested that for clarity and a better understanding of osteomyelitis of the skull the condition be properly classified, especially when one is discussing treatment.

3. The insidious onset, masking the real gravity of the disease, is emphasized.

4. One should never operate on the sinuses unless the indications are clear-cut, but when necessary one should operate promptly and expose adequately for good drainage.

5. One should respect nutrition of the bone and not leave confined pus or necrotic material.

6. One should avoid the use of any instrument that rubs infected material into rough edges of bone, such as the rasp or the curet.

7. The condition should be classified into the localized, or self-limited type, or the progressive type, as soon as possible, so that appropriate treatment may be instituted early.

8. When radical treatment is necessary, one should use it promptly and thoroughly, in stages if necessary.

9. Transfusions should be given preoperatively, postoperatively and intercurrently; the immunotransfusion is especially recommended.

10. Supportive treatment should not be forgotten, regardless of the active treatment.

11. Regeneration of bone in cranial defects is usually good, though it may take several years.

TOBEY.